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NEWS	1		Web Page URLs for STN Seminar Schedule - N. America
NEWS	2		"Ask CAS" for self-help around the clock
NEWS	3	OCT 23	The Derwent World Patents Index suite of databases on STN has been enhanced and reloaded
NEWS	4	OCT 30	CHEMLIST enhanced with new search and display field
NEWS	5	NOV 03	JAPIO enhanced with IPC 8 features and functionality
NEWS	6	NOV 10	CA/CAPLUS F-Term thesaurus enhanced
NEWS	7	NOV 10	STN Express with Discover! free maintenance release Version 8.01c now available
NEWS	8	NOV 20	CAS Registry Number crossover limit increased to 300,000 in additional databases
NEWS	9	NOV 20	CA/CAPLUS to MARPAT accession number crossover limit increased to 50,000
NEWS	10	DEC 01	CAS REGISTRY updated with new ambiguity codes
NEWS	11	DEC 11	CAS REGISTRY chemical nomenclature enhanced
NEWS	12	DEC 14	WPIDS/WPINDEX/WPIX manual codes updated
NEWS	13	DEC 14	GBFULL and FRFULL enhanced with IPC 8 features and functionality
NEWS	14	DEC 18	CA/CAPLUS pre-1967 chemical substance index entries enhanced with preparation role
NEWS	15	DEC 18	CA/CAPLUS patent kind codes updated
NEWS	16	DEC 18	MARPAT to CA/CAPLUS accession number crossover limit increased to 50,000
NEWS	17	DEC 18	MEDLINE updated in preparation for 2007 reload
NEWS	18	DEC 27	CA/CAPLUS enhanced with more pre-1907 records
NEWS	19	JAN 08	CHEMLIST enhanced with New Zealand Inventory of Chemicals
NEWS EXPRESS			NOVEMBER 10 CURRENT WINDOWS VERSION IS V8.01c, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 25 SEPTEMBER 2006.
NEWS HOURS			STN Operating Hours Plus Help Desk Availability
NEWS LOGIN			Welcome Banner and News Items
NEWS IPC8			For general information regarding STN implementation of IPC 8
NEWS X25			X.25 communication option no longer available

Enter NEWS followed by the item number or name to see news on that specific topic.

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\* \* \* \* \* STN Columbus \* \* \* \* \*

FILE 'HOME' ENTERED AT 11:10:17 ON 11 JAN 2007

=> file medline embase biosis caplus  
COST IN U.S. DOLLARS

SINCE FILE ENTRY	TOTAL SESSION
0.42	0.42

FULL ESTIMATED COST

FILE 'MEDLINE' ENTERED AT 11:11:10 ON 11 JAN 2007

FILE 'EMBASE' ENTERED AT 11:11:10 ON 11 JAN 2007  
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FILE 'BIOSIS' ENTERED AT 11:11:10 ON 11 JAN 2007  
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FILE 'CAPLUS' ENTERED AT 11:11:10 ON 11 JAN 2007  
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=> s granulocyte(w)macrophage(w)colony(w)stimulating(w)factor or  
granulocyte(w)colony(w)stimulating(w)factor  
L1 112622 GRANULOCYTE(W) MACROPHAGE(W) COLONY(W) STIMULATING(W) FACTOR OR  
GRANULOCYTE(W) COLONY(W) STIMULATING(W) FACTOR

=> s l1 and (ischemia or hypoxia or Parkinson? or stroke or  
amyotrophic(w)lateral(w)sclerosis or ALS or Lou(w)Gehrig)  
L2 1307 L1 AND (ISCHEMIA OR HYPOXIA OR PARKINSON? OR STROKE OR AMYOTROPH  
IC(W) LATERAL(W) SCLEROSIS OR ALS OR LOU(W) GEHRIG)

=> s l2 and erythropoietin or interleukin  
L3 134 L2 AND ERYTHROPOIETIN OR INTERLEUKIN

=> s l3 and (tpa or tissue(w)plasminogen(w)activator)  
L4 4 L3 AND (TPA OR TISSUE(W) PLASMINOGEN(W) ACTIVATOR)

=> dup rem l4  
PROCESSING COMPLETED FOR L4  
L5 4 DUP REM L4 (0 DUPLICATES REMOVED)

=> dup rem l3  
PROCESSING COMPLETED FOR L3  
L6 113 DUP REM L3 (21 DUPLICATES REMOVED)

=> dis his

(FILE 'HOME' ENTERED AT 11:10:17 ON 11 JAN 2007)

FILE 'MEDLINE, EMBASE, BIOSIS, CAPLUS' ENTERED AT 11:11:10 ON 11 JAN 2007  
L1 112622 S GRANULOCYTE(W)MACROPHAGE(W)COLONY(W)STIMULATING(W)FACTOR OR G  
L2 1307 S L1 AND (ISCHEMIA OR HYPOXIA OR PARKINSON? OR STROKE OR AMYOTR  
L3 134 S L2 AND ERYTHROPOIETIN OR INTERLEUKIN  
L4 4 S L3 AND (TPA OR TISSUE(W)PLASMINOGEN(W)ACTIVATOR)  
L5 4 DUP REM L4 (0 DUPLICATES REMOVED)  
L6 113 DUP REM L3 (21 DUPLICATES REMOVED)

=> dis ibib abs l5

L5 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN  
ACCESSION NUMBER: 2005:1265316 CAPLUS  
DOCUMENT NUMBER: 144:17858  
TITLE: Expression vector with regulatory elements for gene  
expression in mammals

INVENTOR(S): Webster, Keith A.  
 PATENT ASSIGNEE(S): USA  
 SOURCE: U.S. Pat. Appl. Publ., 24 pp., Cont.-in-part of U.S. Ser. No. 723,326.  
 CODEN: USXXCO  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 2  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005266549	A1	20051201	US 2005-118963	20050429
US 6893867	B1	20050517	US 2000-723326	20001128
PRIORITY APPLN. INFO.:			US 1999-171597P	P 19991223
			US 2000-723326	A2 20001128

AB Expression vectors are disclosed that are comprised of (a) one or more silencer elements and conditionally inducible elements to form silencer-inducible regions and (b) promoters in operative linkage upstream of at least one expressed region. The expression vector thereby regulates expression of at least one downstream region by conditional silencing in which an expressed DNA region of a gene is transcribed to produce RNA transcripts, which may or may not be translated to produce polypeptides. Genetically engineered mammalian cells and non-human mammals can be made using such expression vectors through transfection and transgenic techniques. Moreover, processes of making and using the aforementioned products are disclosed (e.g., the expression vector may be used diagnostically, therapeutically, or prophylactically).

=> dis ibib abs l5 2-4

L5 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN  
 ACCESSION NUMBER: 2005:572333 CAPLUS  
 DOCUMENT NUMBER: 143:91472  
 TITLE: Methods of treating neurological conditions with hematopoietic growth factors  
 INVENTOR(S): Schaebitz, Wolf-Ruediger; Schneider, Armin; Krueger, Carola; Sommer, Clemens; Schwab, Stefan; Kollmar, Rainer; Maurer, Martin; Weber, Daniela; Gassler, Nikolaus  
 PATENT ASSIGNEE(S): Axaron Bioscience Ag, Germany  
 SOURCE: U.S. Pat. Appl. Publ., 169 pp., Cont.-in-part of Appl. No. PCT/IB03/06446.  
 CODEN: USXXCO  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 2  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005142102	A1	20050630	US 2004-880101	20040630
US 2004141946	A1	20040722	US 2003-659295	20030911
WO 2004058287	A2	20040715	WO 2003-IB6446	20031231
WO 2004058287	A8	20041021		
WO 2004058287	A3	20041216		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW  
 RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ,

BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE,  
 ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK,  
 TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG  
 WO 2006008582 A1 20060126 WO 2004-IB4329 20041229

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,  
 CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,  
 GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,  
 LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,  
 NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM,  
 SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW  
 RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,  
 IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF,  
 CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM,  
 KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG,  
 KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: US 2002-331755 B1 20021231  
 US 2003-659295 A2 20030911  
 WO 2003-IB6446 A2 20031231  
 US 2004-880101 A 20040630

AB The present invention relates to a method of treating a neurol. condition  
 in a mammal by administering at least one hematopoietic growth factor from  
 the group consisting of GCSF, GMCSF, IL-3, IL-5, a derivative thereof, or a  
 mimetic thereof. A method is also claimed of treating a neurol. condition  
 using neural stem cells treated with a hematopoietic factor. Also claimed  
 is a method of enhancing the survival of a cell transplanted into a  
 mammal, comprising introducing into the cell one or more polynucleotides  
 which encode a hematopoietic factor. A method of enhancing the viability  
 of a neural cell culture comprising contacting the neural cell culture  
 with a hematopoietic factor is addnl. claimed.

L5 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:565109 CAPLUS

DOCUMENT NUMBER: 141:100449

TITLE: Methods of treating neurological conditions with  
 hematopoietic growth factors

INVENTOR(S): Schaebitz, Wolf-Ruediger; Schneider, Armin; Krueger,  
 Carola; Sommer, Clemens; Schwab, Stefan; Kollmar,  
 Rainer; Maurer, Martin; Weber, Daniela; Gassler,  
 Nikolaus

PATENT ASSIGNEE(S): Axaron Bioscience AG, Germany

SOURCE: PCT Int. Appl., 210 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004058287	A2	20040715	WO 2003-IB6446	20031231
WO 2004058287	A8	20041021		
WO 2004058287	A3	20041216		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2004141946	A1	20040722	US 2003-659295	20030911
CA 2511294	A1	20040715	CA 2003-2511294	20031231

AU 2003299430	A1	20040722	AU 2003-299430	20031231
EP 1581249	A2	20051005	EP 2003-799727	20031231
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
BR 2003017910	A	20051129	BR 2003-17910	20031231
CN 1756556	A	20060405	CN 2003-80110075	20031231
JP 2006512419	T	20060413	JP 2005-509731	20031231
US 2005142102	A1	20050630	US 2004-880101	20040630
PRIORITY APPLN. INFO.:			US 2002-331755	A 20021231
			US 2003-659295	A 20030911
			WO 2003-IB6446	W 20031231

AB The present invention relates to a method of treating neurol. conditions in a mammal by administering a hematopoietic growth factor such as granulocyte-colony stimulating factor (GCSF) and granulocyte-macrophage colony stimulating factor (GMCSF). The invention also provides methods of screening for compds. that bind to a GCSF or GMCSF receptor found on the surface of a neuronal cell; and which provides a neuroprotective, neuroproliferative and/or a STAT gene activation activity.

L5 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2003:282718 CAPLUS

DOCUMENT NUMBER: 138:282352

TITLE: Traversal of nucleic acid molecules through a tissue fluid space and expression in repair cells

INVENTOR(S): Sosnowski, Barbara A.; Pierce, Glenn

PATENT ASSIGNEE(S): Selective Genetics, Inc., USA

SOURCE: PCT Int. Appl., 95 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003029429	A2	20030410	WO 2002-US31546	20021002
WO 2003029429	A3	20040401		
WO 2003029429	A9	20040701		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				

AU 2002343475	A1	20030414	AU 2002-343475	20021002
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US 2003148979	A1	20030807	US 2002-264284	20021002
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EP 1438413	A2	20040721	EP 2002-780419	20021002
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R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK

PRIORITY APPLN. INFO.: US 2001-327513P P 20011003

WO 2002-US31546 W 20021002

AB Disclosed are methods for use in transferring nucleic acids into cells at a wound site associated with a fluid space. These gene transfer protocols are suitable for use in transferring various nucleic acids into cartilage, cardiac muscle, and other tissues, and have many uses including treating diseases such as arthritis and ischemic heart disease, and promoting wound healing. The invention further disclosed pharmaceutical compns. that may be used in the practice of the invention to transfer the nucleic acid of

interest. Such compns. include any multi-partitioned biocompatible matrix in combination with multiple nucleic acids of interest. Thus, collagen collagen-immobilized fibroblast growth factor (FGF) genes induce angiogenesis in vitro, and FGF gene delivery to skeletal muscle wounds induces both angiogenesis and arteriogenesis and well as induces myocyte regeneration.

=> dis his

(FILE 'HOME' ENTERED AT 11:10:17 ON 11 JAN 2007)

FILE 'MEDLINE, EMBASE, BIOSIS, CAPLUS' ENTERED AT 11:11:10 ON 11 JAN 2007

L1 112622 S GRANULOCYTE(W)MACROPHAGE(W)COLONY(W)STIMULATING(W)FACTOR OR G  
L2 1307 S L1 AND (ISCHEMIA OR HYPOXIA OR PARKINSON? OR STROKE OR AMYOTR  
L3 134 S L2 AND ERYTHROPOIETIN OR INTERLEUKIN  
L4 4 S L3 AND (TPA OR TISSUE(W)PLASMINOGEN(W)ACTIVATOR)  
L5 4 DUP REM L4 (0 DUPLICATES REMOVED)  
L6 113 DUP REM L3 (21 DUPLICATES REMOVED)

=> dis ibib abs l6 100-113

L6 ANSWER 100 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 96235644 EMBASE

DOCUMENT NUMBER: 1996235644

TITLE: Possible role of tumor necrosis factor-alpha in erythropoietic suppression by endotoxin and granulocyte/macrophage colony-stimulating factor.

AUTHOR: Udupa K.B.; Sharma B.G.

CORPORATE SOURCE: Dr. K.B. Udupa, VA Medical Center, 4300 West Seventh Street, Little Rock, AR 72205, United States

SOURCE: American Journal of Hematology, (1996) Vol. 52, No. 3, pp. 178-183.

ISSN: 0361-8609 CODEN: AJHEDD

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 025 Hematology

026 Immunology, Serology and Transplantation

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 18 Jun 1997

Last Updated on STN: 18 Jun 1997

AB Injection of bacterial endotoxin or granulocyte/macrophage colony-stimulating factor (GM-CSF) into exhypoxic polycythemic mice simultaneously with erythropoietin (EPO) suppressed erythroid cell formation, as monitored by <sup>59</sup>Fe incorporation into circulating red blood cells. This effect was dose-dependent and time-dependent, GM-CSF did not inhibit erythroid cell formation directly, as the antibody to the GM-CSF did not neutralize the effect of endotoxin, the inducer of GM-CSF. The suppression of both agents could be partially corrected by prior injection of a monoclonal antibody to tumor necrosis factor  $\alpha$  (anti-TNF $\alpha$ ). These results indicate that the suppression of EPO-induced erythroid cell formation by endotoxin and GM-CSF was due in part to the production of TNF $\alpha$ .

L6 ANSWER 101 OF 113 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 1996:440012 BIOSIS

DOCUMENT NUMBER: PREV199699162368

TITLE: Biology and pathophysiology of leukotrienes.

AUTHOR(S): Denzlinger, Claudio

CORPORATE SOURCE: Med. Klinik III, Klinikum Grosshadern, Ludwig-Maximilians  
Univ. Muenchen, 81377 Muenchen, Germany  
SOURCE: Critical Reviews in Oncology-Hematology, (1996) Vol. 23,  
No. 3, pp. 167-223.  
ISSN: 1040-8428.  
DOCUMENT TYPE: Article  
General Review; (Literature Review)  
LANGUAGE: English  
ENTRY DATE: Entered STN: 7 Oct 1996  
Last Updated on STN: 7 Oct 1996

L6 ANSWER 102 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights  
reserved on STN

ACCESSION NUMBER: 96158763 EMBASE  
DOCUMENT NUMBER: 1996158763  
TITLE: Potential role of hemopoietic cytokines in neuronal  
survival.  
AUTHOR: Sigel K.; Rosenbaum D.M.  
CORPORATE SOURCE: Dept. of Neurology, Albert Einstein College of Medicine,  
1300 Morris Park Ave., Bronx, NY 10461, United States  
SOURCE: Drug News and Perspectives, (1996) Vol. 9, No. 3, pp.  
142-148.  
ISSN: 0214-0934 CODEN: DNPEED  
COUNTRY: Spain  
DOCUMENT TYPE: Journal; General Review  
FILE SEGMENT: 002 Physiology  
005 General Pathology and Pathological Anatomy  
008 Neurology and Neurosurgery  
025 Hematology  
029 Clinical Biochemistry  
030 Pharmacology  
037 Drug Literature Index  
LANGUAGE: English  
ENTRY DATE: Entered STN: 3 Jul 1996  
Last Updated on STN: 3 Jul 1996

DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L6 ANSWER 103 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights  
reserved on STN

ACCESSION NUMBER: 95334463 EMBASE  
DOCUMENT NUMBER: 1995334463  
TITLE: Treatment with retinoids and haemopoietic growth factors in  
myelodysplastic syndromes [1].  
AUTHOR: Ciotti R.; Rosti V.; Lucotti C.; Forloni F.; Romeo G.;  
Pezzoli A.  
CORPORATE SOURCE: First Div. of Internal Medicine, Ospedale Consorziiale,  
Azienda Sanitaria 13, 24047 Treviglio, Italy  
SOURCE: British Journal of Haematology, (1995) Vol. 91, No. 3, pp.  
773-774.  
ISSN: 0007-1048 CODEN: BJHEAL  
COUNTRY: United Kingdom  
DOCUMENT TYPE: Journal; Letter  
FILE SEGMENT: 016 Cancer  
025 Hematology  
037 Drug Literature Index  
038 Adverse Reactions Titles  
LANGUAGE: English  
ENTRY DATE: Entered STN: 5 Dec 1995  
Last Updated on STN: 5 Dec 1995

DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L6 ANSWER 104 OF 113 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on  
STN

ACCESSION NUMBER: 1995:505151 BIOSIS

DOCUMENT NUMBER: PREV199598510201  
 TITLE: Erythropoietin and deferred autologous blood collection: From physiological secretion to the rationale for exogenous supplementation.  
 AUTHOR(S): Casadevall, N.  
 CORPORATE SOURCE: Hop. Raymond Poincare, Lab. Hematol., 104 blvd. Raymond Poincare, F-92380 Garches, France  
 SOURCE: Nouvelle Revue Francaise d'Hematologie, (1995) Vol. 37, No. SUPPL. 1, pp. S11-S15.  
 CODEN: NRFHA4. ISSN: 0029-4810.  
 DOCUMENT TYPE: Article  
 General Review; (Literature Review)  
 LANGUAGE: French  
 ENTRY DATE: Entered STN: 29 Nov 1995  
 Last Updated on STN: 29 Nov 1995

L6 ANSWER 105 OF 113 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1994:699076 CAPLUS  
 DOCUMENT NUMBER: 121:299076  
 TITLE: The kinetoplastid membrane protein 11 of Leishmania donovani and African trypanosomes is a potent stimulator of T-lymphocyte proliferation  
 AUTHOR(S): Tolson, Douglas L.; Jardim, Armando; Schnur, Lionel F.; Stebeck, Caroline; Tuckey, Corinna; Beecroft, Robert P.; Teh, Hung-Sia; Olafson, Robert W.; Pearson, Terry W.  
 CORPORATE SOURCE: Dep. Biochem. Microbiol., Univ. Victoria, Victoria, BC, V8W 3P6, Can.  
 SOURCE: Infection and Immunity (1994), 62(11), 4893-9  
 CODEN: INFIBR; ISSN: 0019-9567  
 PUBLISHER: American Society for Microbiology  
 DOCUMENT TYPE: Journal  
 LANGUAGE: English

AB Kinetoplastid membrane protein 11 (KMP-11) from Leishmania donovani is an abundant 11-kDa surface membrane glycoprotein. Lymph node cells from mice of six different H-2 haplotypes immunized with KMP-11 or with L. donovani promastigotes were stimulated to proliferate in vitro with purified KMP-11. Primed purified T cells required antigen presentation since they were not stimulated unless KMP-11-pulsed or L. donovani-infected macrophages were added. Promastigotes of a wide variety of Leishmania species and procyclic forms of African trypanosomes stimulated proliferation of KMP-11-primed or L. donovani promastigote-primed lymph node cells. All of the Leishmania promastigotes and African trypanosomes tested contained an 11-kDa protein, as detected by immunoblotting with KMP-11-specific monoclonal antibodies. The widespread distribution of the 11-kDa (KMP-11) mols. and their ability to stimulate strong T-lymphocyte proliferation in a non-H-restricted fashion suggest that they may be important mols. for induction of cell-mediated immune responses.

L6 ANSWER 106 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 94115823 EMBASE  
 DOCUMENT NUMBER: 1994115823  
 TITLE: Evidence suggesting a negative regulatory role for macrophages in murine erythropoiesis in vivo.  
 AUTHOR: Wang C.Q.; Udupa K.B.; Xiao H.; Lipschitz D.A.  
 CORPORATE SOURCE: John L. McClelland, Memorial Veterans Hospital, 4300 West Seventh Street, Little Rock, AR 72205, United States  
 SOURCE: Experimental Hematology, (1994) Vol. 22, No. 4, pp. 370-376.  
 ISSN: 0301-472X CODEN: EXHEBH  
 COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article  
 FILE SEGMENT: 025 Hematology



## 037 Drug Literature Index

LANGUAGE: English  
SUMMARY LANGUAGE: English  
ENTRY DATE: Entered STN: 25 May 1994  
Last Updated on STN: 25 May 1994

AB Increasing the rate of erythropoiesis in C57BL/6 mice, either by hypoxia or by the injection of recombinant erythropoietin (Epo), resulted in significant reductions in marrow macrophage number, as assessed by flow cytometry employing the monoclonal antibody against the macrophage antigen Mac-1 and by histologic determination of reductions in the number of marrow esterase-positive cells. This decline was paralleled by decreases in marrow colony-forming unit-macrophage (CFU-M) and colony-forming unit-granulocyte/macrophage (CFU-GM) number. The intramedullary concentration of the cytokines interleukin-1 $\alpha$  (IL-1 $\alpha$ ) and tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ), which are produced by macrophages, was also reduced. Cessation of erythropoiesis was associated with increases in macrophage number, CFU-M and CFU-GM colony number, and IL-1 $\alpha$  concentrations. Increased erythropoiesis resulted in reductions in number of burst-forming unit-erythroid (BFU-E) colonies, which were less sensitive to suppression by macrophages as evidenced by less increase in colony number when macrophages were removed from the marrow before in vitro BFU-E culture. BFU-E colony number was suppressed less when IL-1 $\alpha$  and TNF- $\alpha$  were added to cultures obtained from animals with stimulated erythropoiesis. Compared to controls, BFU-E number and suppression by macrophages increased significantly when erythropoiesis was reduced. These observations provide compelling evidence for a regulatory role for macrophages in normal erythropoiesis in vivo, presumably acting as a negative balance to the stimulatory effects of Epo.

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ACCESSION NUMBER: 94010380 EMBASE  
DOCUMENT NUMBER: 1994010380  
TITLE: Recombinant human DNase for treatment of cystic fibrosis.  
AUTHOR: Wordell C.J.  
CORPORATE SOURCE: Drug Information Service, Department of Pharmacy, Thomas Jefferson University Hospital, 11th and Walnut Streets, Philadelphia, PA 19107, United States  
SOURCE: Hospital Pharmacy, (1993) Vol. 28, No. 12, pp. 1226+1229-1232+1240. .  
ISSN: 0018-5787 CODEN: HOPHAZ  
COUNTRY: United States  
DOCUMENT TYPE: Journal; Note  
FILE SEGMENT: 006 Internal Medicine  
015 Chest Diseases, Thoracic Surgery and Tuberculosis  
037 Drug Literature Index  
LANGUAGE: English  
ENTRY DATE: Entered STN: 30 Jan 1994  
Last Updated on STN: 30 Jan 1994

DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L6 ANSWER 108 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 93080744 EMBASE  
DOCUMENT NUMBER: 1993080744  
TITLE: [Hematopoietic growth hormone factors as adjuncts in antiretroviral therapy].  
HAMATOPOETISCHE WACHSTUMSFAKTOREN ALS  
ZUSATZBEHANDLUNG BEI DER ANTIRETROVIRALEN THERAPIE.  
SOURCE: AIDS-Forschung, (1993) Vol. 8, No. 2, pp. 69-70. .  
ISSN: 0179-3098 CODEN: AIFOER  
COUNTRY: Germany  
DOCUMENT TYPE: Journal; Note

FILE SEGMENT: 004 Microbiology  
025 Hematology  
026 Immunology, Serology and Transplantation  
037 Drug Literature Index  
038 Adverse Reactions Titles

LANGUAGE: German

ENTRY DATE: Entered STN: 18 Apr 1993  
Last Updated on STN: 18 Apr 1993

DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L6 ANSWER 109 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 93073784 EMBASE  
DOCUMENT NUMBER: 1993073784  
TITLE: Stimulating new developments: Colony-stimulating factors.  
AUTHOR: Kare D.  
CORPORATE SOURCE: Home Nutritional Services, Chicago, IL, United States  
SOURCE: Journal of Intravenous Nursing, (1993) Vol. 16, No. 1, pp. 37-43.

ISSN: 0896-5846 CODEN: JINUEE

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 025 Hematology  
037 Drug Literature Index  
038 Adverse Reactions Titles

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 11 Apr 1993  
Last Updated on STN: 11 Apr 1993

AB Drug-induced low white-blood-cell counts have long impaired our ability to treat patients. Colony-stimulating factors are now available for intravenous or subcutaneous administration. These glycoproteins act on hematopoietic cells by binding to specific cell surface receptors and by stimulating proliferation, differentiation, commitment, and activation of new white blood cells. A brief overview of patient population, indications, actions, and adverse reactions for hospital or home use is presented.

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ACCESSION NUMBER: 92284673 EMBASE  
DOCUMENT NUMBER: 1992284673  
TITLE: [Growth factors in hematology].  
GROEIFACTOREN IN DE HEMATOLOGIE.  
AUTHOR: Demyunck H.; Boogaerts M.A.  
CORPORATE SOURCE: Afdeling Hematologie, Universitaire Ziekenhuizen, Katholieke Universiteit, Leuven, Belgium  
SOURCE: Tijdschrift voor Geneeskunde, (1992) Vol. 48, No. 16, pp. 1187-1196.

ISSN: 0371-683X CODEN: TGEKBW

COUNTRY: Belgium

DOCUMENT TYPE: Journal; (Short Survey)

FILE SEGMENT: 016 Cancer  
025 Hematology  
026 Immunology, Serology and Transplantation  
029 Clinical Biochemistry  
030 Pharmacology  
037 Drug Literature Index  
038 Adverse Reactions Titles

LANGUAGE: Dutch

ENTRY DATE: Entered STN: 25 Oct 1992  
Last Updated on STN: 25 Oct 1992

DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L6 ANSWER 111 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 92343779 EMBASE  
DOCUMENT NUMBER: 1992343779  
TITLE: Regulation of erythropoiesis in the newborn: A complex system.  
AUTHOR: Heikinheimo M.; Siimes M.A.  
CORPORATE SOURCE: The Children's Hospital, University of Helsinki, SF-00290 Helsinki, Finland  
SOURCE: Annals of Medicine, (1992) Vol. 24, No. 5, pp. 309-311. .  
ISSN: 0785-3890 CODEN: ANMDEU  
COUNTRY: United Kingdom  
DOCUMENT TYPE: Journal; Editorial  
FILE SEGMENT: 007 Pediatrics and Pediatric Surgery  
010 Obstetrics and Gynecology  
021 Developmental Biology and Teratology  
025 Hematology  
029 Clinical Biochemistry  
037 Drug Literature Index  
LANGUAGE: English  
ENTRY DATE: Entered STN: 13 Dec 1992  
Last Updated on STN: 13 Dec 1992  
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER:

L6 ANSWER 112 OF 113 MEDLINE on STN DUPLICATE 10  
ACCESSION NUMBER: 91358449 MEDLINE  
DOCUMENT NUMBER: PubMed ID: 1653242  
TITLE: Hypoxia up-regulates the activity of a novel erythropoietin mRNA binding protein.  
AUTHOR: Rondon I J; MacMillan L A; Beckman B S; Goldberg M A; Schneider T; Bunn H F; Malter J S  
CORPORATE SOURCE: Department of Pharmacology, Tulane University School of Medicine, New Orleans, Louisiana 70112.  
CONTRACT NUMBER: CA-01427 (NCI)  
DK-01401 (NIDDK)  
DK-41234 (NIDDK)  
SOURCE: The Journal of biological chemistry, (1991 Sep 5) Vol. 266, No. 25, pp. 16594-8.  
Journal code: 2985121R. ISSN: 0021-9258.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 199110  
ENTRY DATE: Entered STN: 27 Oct 1991  
Last Updated on STN: 3 Feb 1997  
Entered Medline: 4 Oct 1991

AB The mechanisms which control the production of erythropoietin (Epo) remain enigmatic. Recent data suggest that the half-time of Epo messenger RNA (mRNA) is increased by hypoxia in Hep 3B cells, a human hepatoma line. The post-transcriptional regulation of other rapidly degraded mRNAs is mediated by sequence-specific mRNA binding proteins. In order to determine if Epo mRNA specific binding proteins exist, we probed cytosolic lysates from Hep 3B cells and mouse tissues with radiolabeled Epo RNA. A cytosolic protein that binds specifically to Epo RNA was identified in the Epo-producing, hepatoblastoma Hep 3B cell line by gel mobility shift assay. This protein was identified in both normoxic and hypoxic cells and bound specifically to a 120-base fragment of the 3'-untranslated region (3'-UTR) of Epo mRNA. Binding was completed with unlabeled Epo RNA, but not with granulocyte-macrophage colony-stimulating factor RNA. Ultraviolet light cross-linked Epo RNA-protein complexes migrated as two bands of 70 and 135-140 kD on sodium dodecyl sulfate-polyacrylamide gels. Binding activity was markedly increased in brain and spleen lysates from mice

subjected to 24 h of hypoxia. Therefore, the post-transcriptional regulation of Epo expression in response to hypoxia may in part be due to the interaction of Epo RNA with its specific binding protein.

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ACCESSION NUMBER: 88272110 EMBASE  
DOCUMENT NUMBER: 1988272110  
TITLE: Up-regulation of interleukin 4/B-cell stimulatory factor 1 receptor expression.  
AUTHOR: Ohara J.; Paul W.E.  
CORPORATE SOURCE: Laboratory of Immunology, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD 20892, United States  
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1988) Vol. 85, No. 21, pp. 8221-8225.  
ISSN: 0027-8424 CODEN: PNASA6  
COUNTRY: United States  
DOCUMENT TYPE: Journal  
FILE SEGMENT: 026 Immunology, Serology and Transplantation  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
ENTRY DATE: Entered STN: 11 Dec 1991  
Last Updated on STN: 11 Dec 1991

AB The expression of interleukin 4 (IL-4) receptors on resting T and B lymphocytes was enhanced 4- to 8-fold by IL-4 stimulation of these cells. Other agents such as lipopolysaccharide and anti-IgM for B cells and concanavalin A for T cells also caused increased IL-4 receptor expression, although to a somewhat smaller degree than IL-4. Using a newly developed flow cytometric analysis based on the binding of biotinylated IL-4 and phycoerythrin-streptavidin, it was observed that receptor up-regulation in a T-cell population treated with IL-4 was a feature of the majority of the T cells. Analysis of IL-4 by cross-linkage of 125I-labeled IL-4 to IL-4 receptor with disuccinimidyl suberate indicated that the IL-4-IL-4 receptor complex was the same size in the resting and up-regulated cells, implying that the same receptor species found in resting cells was up-regulated in response to IL-4.

=> dis ibib abs l6 90-99

L6 ANSWER 90 OF 113 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 1999:131347 BIOSIS  
DOCUMENT NUMBER: PREV199900131347  
TITLE: Peptides as drugs.  
AUTHOR(S): Edwards, C. M. B.; Cohen, M. A.; Bloom, S. R.  
CORPORATE SOURCE: ICSM Endocrine Unit, Hammersmith Hosp., London, UK  
SOURCE: QJM, (Jan., 1999) Vol. 92, No. 1, pp. 1-4. print.  
CODEN: QJMEA7. ISSN: 0033-5622.  
DOCUMENT TYPE: Article  
Editorial  
LANGUAGE: English  
ENTRY DATE: Entered STN: 17 Mar 1999  
Last Updated on STN: 17 Mar 1999

L6 ANSWER 91 OF 113 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1998:706077 CAPLUS  
DOCUMENT NUMBER: 129:321206  
TITLE: Sustained-release alginate gels  
INVENTOR(S): Goldenberg, Merrill Seymour; Beekman, Alice C.  
PATENT ASSIGNEE(S): Amgen Inc., USA

SOURCE: PCT Int. Appl., 41 pp.  
 CODEN: PIXXD2  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 1  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9846211	A1	19981022	WO 1998-US7566	19980414
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 2002001619	A1	20020103	US 1997-842756	19970417
US 6656508	B2	20031202		
ZA 9803089	A	19981019	ZA 1998-3089	19980414
CA 2286092	A1	19981022	CA 1998-2286092	19980414
CA 2286092	C	20041214		
AU 9869734	A	19981111	AU 1998-69734	19980414
EP 975333	A1	20000202	EP 1998-915592	19980414
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2001524084	T	20011127	JP 1998-544247	19980414
TW 577755	B	20040301	TW 1998-87105641	19980414
MX 9909388	A	20000630	MX 1999-9388	19991013

PRIORITY APPLN. INFO.:  
 US 1997-842756 A 19970417  
 WO 1998-US7566 W 19980414

AB The present invention relates to sustained-release formulations using alginate gel beads. Small alginate beads were prepared by using 25mM ZnCl<sub>2</sub> in the bath. As the concentration of the leptin in the bead increased, the fractional release of leptin from the bead decreased.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 92 OF 113 CAPLUS COPYRIGHT 2007 ACS on STN  
 ACCESSION NUMBER: 1998:323165 CAPLUS  
 DOCUMENT NUMBER: 129:8577  
 TITLE: Methods for regulating angiogenesis  
 INVENTOR(S): Isner, Jeffrey M.; Asahara, Takayuki  
 PATENT ASSIGNEE(S): St. Elizabeth's Medical Center of Boston, Inc., USA  
 SOURCE: PCT Int. Appl., 57 pp.  
 CODEN: PIXXD2  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 2  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9819712	A1	19980514	WO 1997-US19935	19971106
W: AU, CA, JP				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 5980887	A	19991109	US 1996-744882	19961108
CA 2271690	A1	19980514	CA 1997-2271690	19971106
AU 9852432	A	19980529	AU 1998-52432	19971106
AU 743267	B2	20020124		
EP 941125	A1	19990915	EP 1997-947319	19971106
R: DE, FR, GB, IT				
JP 2001503427	T	20010313	JP 1998-521645	19971106

EP 1618898                      A2            20060125            EP 2005-17496                      19971106  
 R: DE, FR, GB, IT  
 PRIORITY APPLN. INFO.:                      US 1996-744882            A 19961108  
    EP 1997-947319            A3 19971106  
    WO 1997-US19935            W 19971106

AB    In accordance with the present invention, EC (endothelial cell) progenitors can be used in a method for regulating angiogenesis, i.e., enhancing or inhibiting blood vessel formation, in a selected patient and in some preferred embodiments for targetting specific locations. For example, the EC progenitors can be used to enhance angiogenesis or to deliver an angiogenesis modulator, e.g. anti- or pro-angiogenic agents, resp. to sites of pathol. or utilitarian angiogenesis. Addnl., in another embodiment, EC progenitors can be used to induce reendothelialization of an injured blood vessel, and thus reduce restenosis by indirectly inhibiting smooth muscle cell proliferation.

REFERENCE COUNT:                      11            THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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ACCESSION NUMBER:    1998320520    EMBASE  
 TITLE:                      Successful treatment of a Jehovah's witness with acute promyelocytic leukemia by all-trans retinoic acid.  
 AUTHOR:                      Kajiguchi T.; Yamamoto Y.; Miyata Y.; Saito M.; Takeyama H.  
 CORPORATE SOURCE:    Dr. T. Kajiguchi, Department of Internal Medicine, Nagoya Ekisaikai Hospital, 4-66 Shonen-cho, Nakagawa-ku, Nagoya 454-8502, Japan  
 SOURCE:                      Biotherapy, (1998) Vol. 12, No. 8, pp. 1159-1163. .  
    Refs: 7  
    ISSN: 0914-2223    CODEN: BITPE  
 COUNTRY:                      Japan  
 DOCUMENT TYPE:                      Journal; Article  
 FILE SEGMENT:                      005            General Pathology and Pathological Anatomy  
    006            Internal Medicine  
    016            Cancer  
    025            Hematology  
    037            Drug Literature Index  
 LANGUAGE:                      Japanese  
 SUMMARY LANGUAGE:    English; Japanese  
 ENTRY DATE:                      Entered STN: 15 Oct 1998  
    Last Updated on STN: 15 Oct 1998

AB    A 23-year-old woman was referred to our hospital for treatment of acute promyelocytic leukemia (APL). She was a Jehovah's Witness and would not accept blood products. The Ethics Committee of our hospital recommended accommodation of her religious beliefs, because the patient and her family thoroughly understood and accepted the increased risk of fatal bleeding and severe hypoxia due to anemia during the therapy without using blood products. After the patient signed a special consent form, we began treatment with all- trans retinoic acid (ATRA, 45 mg/m2 daily). She also received erythropoietin and granulocyto colony-stimulating factor. Sixty days after administration of ATRA, she achieved a complete remission (CR). After CR she received 3 cycles of chemotherapy for consolidation, which is according to the protocol of the Japan Adult Leukemia Study Group (AML-92). During the therapy, the patient did not receive blood products. No PML-RARA amplification products were detected by the reverse transcriptase polymerase chain reaction in her bone marrow at the end of the chemotherapy Blood product support is usually requisite for standard chemotherapy for APL because of the bleeding tendency. ATRA can induce differentiation of the leukemic clone without myelo-suppression, so we can treat APL patients without using blood products.

L6    ANSWER 94 OF 113    EMBASE    COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER:    1998359165    EMBASE

TITLE: Strategies for the use of epoetin alfa in breast cancer patients.  
 AUTHOR: Del Mastro L.; Venturini M.  
 CORPORATE SOURCE: Dr. L. Del Mastro, Oncologia Medica 1, Ist. Nazionale Ricerca Cancro, L. go Rosanna Benzi 10, 16132 Genova, Italy. mventur@hp380.ist.unige.it  
 SOURCE: Oncologist, (1998) Vol. 3, No. 5, pp. 314-318. .  
 Refs: 27  
 ISSN: 1083-7159 CODEN: OCOLF6  
 COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article  
 FILE SEGMENT: 016 Cancer  
 025 Hematology  
 030 Pharmacology  
 037 Drug Literature Index  
 038 Adverse Reactions Titles  
 LANGUAGE: English  
 SUMMARY LANGUAGE: English  
 ENTRY DATE: Entered STN: 19 Nov 1998  
 Last Updated on STN: 19 Nov 1998

AB Anemia is a common complication in cancer patients undergoing chemotherapy, and its severity depends on both the type of antineoplastic drugs and the clinical status of the patient. Breast cancer patients undergoing standard chemotherapy develop clinically significant anemia in up to 25% of cases. This percentage, moreover, increases up to 63% when more intensive chemotherapy regimens are used. The therapeutic use of erythropoietin in anemic patients, i.e., in patients with hemoglobin levels below 9-10.5 g/dl, is able to correct the anemic status in nearly 40%-80% of such patients, but it does not completely eliminate the need of blood transfusions: 20%-40% of patients need to be transfused despite the erythropoietin treatment. An alternative strategy for optimizing the erythropoietin treatment is its use in the prevention of anemia, i.e., in patients with normal hemoglobin values but at high risk of becoming anemic. In a phase III study, we evaluated the role of erythropoietin in the prevention of anemia in breast cancer patients undergoing dose-intensive chemotherapy. Clinically significant anemia occurred in 52% (95% CI = 33-69) of control patients and in no patient (95% CI = 0-14) in the erythropoietin arm ( $p = .00001$ ). After six cycles of chemotherapy the mean hemoglobin decrease was 3.05 g/dl ( $\pm 1.0$ , 95% CI = 2.6-3.5) in the control arm and 0.8 g/dl ( $\pm 1.4$ , 95% CI = 0.3-1.4) in the erythropoietin arm. Moreover, 6.4% of control patients needed blood transfusion compared to no patients in the erythropoietin arm. Erythropoietin is active in both the treatment and the prevention of anemia in cancer patients undergoing chemotherapy. Due to its high economic cost, efforts should be made to identify subsets of patients in whom the preventive use could be cost-effective. Patients undergoing chemotherapy associated with a high risk of anemia could benefit from preventive use of erythropoietin in special circumstances, such as presence of risk of myocardial or cerebral ischemia, uncommon blood group, or religious beliefs hindering blood transfusions. Moreover, anemia prevention could be considered in patients at high risk of requiring blood transfusions, such as patients with low baseline value of hemoglobin or with a hemoglobin decrease of  $\geq 2$  g/dl after the first cycle of chemotherapy.

L6 ANSWER 95 OF 113 MEDLINE on STN DUPLICATE 9  
 ACCESSION NUMBER: 1998140501 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 9479872  
 TITLE: Markedly high plasma erythropoietin and granulocyte-colony stimulating factor levels in patients with paroxysmal nocturnal hemoglobinuria.  
 AUTHOR: Nakakuma H; Nagakura S; Kawaguchi T; Horikawa K; Iwamoto N; Kagimoto T; Takatsuki K

CORPORATE SOURCE: Second Department of Internal Medicine, Kumamoto University  
School of Medicine, Japan.  
SOURCE: International journal of hematology, (1997 Dec) Vol. 66,  
No. 4, pp. 451-7.  
Journal code: 9111627. ISSN: 0925-5710.  
PUB. COUNTRY: Ireland  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 199803  
ENTRY DATE: Entered STN: 26 Mar 1998  
Last Updated on STN: 26 Mar 1998  
Entered Medline: 17 Mar 1998

AB In patients with paroxysmal nocturnal hemoglobinuria (PNH), we measured plasma concentrations of endogenous hematopoiesis-regulatory cytokines to characterize bone marrow (BM) hypoplasia which is a major cause of death. Contrary to 10 healthy individuals, all 14 patients with PNH showed increases of erythropoietin (Epo) and granulocyte-colony stimulating factor (G-CSF). There were no signs of infection, renal dysfunction or hypoxia. The lower the hemoglobin level and granulocyte count, the higher the plasma Epo and G-CSF levels. In contrast, marked differences were not found in the levels of interleukin-3 (IL-3), tumor necrosis factor-alpha (TNF-alpha), stem cell factor (SCF), granulocyte/macrophage-colony stimulating factor (GM-CSF), or interferon-gamma (IF-gamma). The cytokine profiles of PNH patients were quite similar to those of patients with aplastic anemia (AA) and myelodysplastic syndrome (MDS). The cytokine profiles may support a pathological relationship between PNH and these stem cell disorders.

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ACCESSION NUMBER: 1997:199747 BIOSIS  
DOCUMENT NUMBER: PREV199799498950  
TITLE: Hematology of the elderly patient: The red series: Anemia of the elderly patient.  
AUTHOR(S): Florez-Tascon Sixto, F. J.; Sanchez-Escribano, F.; Siguin Gomez, A.; Herraez, R.; Cobos, J.; Ruiz Martin, J.  
SOURCE: Geriatrika (Madrid), (1997) Vol. 13, No. 1, pp. 17-21.  
ISSN: 0212-9744.  
DOCUMENT TYPE: Article  
LANGUAGE: Spanish  
ENTRY DATE: Entered STN: 12 May 1997  
Last Updated on STN: 12 May 1997

L6 ANSWER 97 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 97330591 EMBASE  
DOCUMENT NUMBER: 1997330591  
TITLE: [Erythropoietin: Biochemical profile, biological records, indications and therapeutic results in hematology].  
ERITROPOIETINA: PROFILO BIOCHIMICO, RICORDI BIOLOGICI, INDICAZIONI E RISULTATI TERAPEUTICI IN EMATOLOGIA.  
AUTHOR: Marmont A.M.  
CORPORATE SOURCE: Prof. A.M. Marmont, II Divisione di Ematologia, Ospedale San Martino, Piazzale R. Benzi 10, 16132 Genova, Italy  
SOURCE: Tumori, (1997) Vol. 83, No. 4 SUPPL. 2, pp. S3-S15. .  
Refs: 146  
ISSN: 0300-8916 CODEN: TUMOAB  
COUNTRY: Italy  
DOCUMENT TYPE: Journal; Conference Article  
FILE SEGMENT: 016 Cancer  
025 Hematology



029 Clinical Biochemistry  
037 Drug Literature Index

LANGUAGE: Italian  
SUMMARY LANGUAGE: English  
ENTRY DATE: Entered STN: 1 Dec 1997

Last Updated on STN: 1 Dec 1997

AB This review has two objects: a brief recapitulation of the biological background of erythropoietin (EPO), and a review of its clinical utilization in hematology. EPO, both in its naturally occurring and recombinant form (rH-EPO), is a single chain glycoprotein with an approximate molecular weight of 30,000 to 34,000 kD. Its heavy glycosylation is essential for its activity in vivo, since asialoEPO is readily cleared by the hepatic asialoglycoprotein receptor. This impedes the recombinant molecule's synthesis in biologic cultures other than mammalian cells (Chinese hamster's ovary cells), and inevitably increases costs. If in vitro glycosylation of E. coli-derived rH-EPO could be achieved, the clinical utilization of the product would be considerably enhanced, most especially when very high doses are necessary, as discussed later. There is no antigenic diversity between natural and recombinant EPO, so that out of the enormous clinical experience only one single case of immunization has been recorded. Almost paradoxically there are however three published cases of pure red cell aplasia (PRCA) caused by immunization against autologous EPO. It is now established that in adults EPO is synthesized in renal peritubular interstitial cells, although some residual activity remains in the liver. Hypoxia results in a rapid induction of EPO expression, although the role of the oxygen sensor system is still debated. Cellular targets are notoriously erythroid progenitors and precursors (BFU-E, CFU-E, early and intermediate erythroblasts). The global erythropoietic activity resulted in various effects (proliferation, differentiation, survival), but most probably each single effect is integrated with and complementary of the others. The utilization of rH-EPO in hematologic diseases came much later than its dramatic success in renal anemia. A variety of tools useful for assessing the possible beneficial effects of rH-EPO in clinical hematology has been proposed, among which a low level of endogenous EPO is a good predictor for therapeutic success. 'Hemopathic' anemia can be subdivided into three categories: patients with normal erythropoiesis due to inadequate EPO production (anemia of prematurity), patients with depressed but nonclonal erythropoiesis (chemotherapy, lymphoid malignancies such as multiple myeloma MM and chronic lymphatic leukemia - CLL) and patients with at least partially clonal anemia, such as paroxysmal nocturnal hemoglobinuria (PNH), hemoglobinopathies, myelodysplastic syndromes (MDS) and others. Results in the first category of patients are, as expected, prompt and satisfactory with physiologic doses. Although therapeutic strategy for MM is moving fast to curative intents, the utilization of rH-EPO is indicated or the control of anemia in conservatively-treated patients. In the third category the most important and controversial area is MDS. Significant erythropoietic results are generally obtained in about 20% of patients; however, the association with G-CSF has considerably enhanced the response rate. In the field of bone marrow transplantation there is an inadequate production of endogenous EPO in the allogeneic setting, and randomized studies have shown the benefits of rH-EPO in this situation. However, the most important results have been and are obtained in post-major-ABO incompatible PRCA, when the removal of the recipient's isohemagglutinins does not resolve the anemia. High and very high doses of rH-EPO (even over 500 U/kg/day for 2-4 weeks) may resolve this occasionally quite refractory condition. Although extremely expensive, this treatment may be life-saving when an otherwise successful allogeneic transplant is at the risk of failure because of this relatively uncommon but severe immunohematologic complication.

L6 ANSWER 98 OF 113 CAPLUS COPYRIGHT 2007 ACS on STN  
ACCESSION NUMBER: 1996:756546 CAPLUS  
DOCUMENT NUMBER: 126:17804

TITLE: Human antibodies derived from immunized xenomice  
 INVENTOR(S): Kucherlapati, Raju; Jakobovits, Aya; Klapholz, Sue;  
 Brenner, Daniel G.; Capon, Daniel J.  
 PATENT ASSIGNEE(S): Cell Genesys, Inc., USA  
 SOURCE: PCT Int. Appl., 64 pp.  
 CODEN: PIXXD2  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 1  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9634096	A1	19961031	WO 1995-US5500	19950428
W: AU, CA, FI, HU, JP, KR, NO, NZ				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
CA 2219486	A1	19961031	CA 1995-2219486	19950428
AU 9524668	A	19961118	AU 1995-24668	19950428
EP 823941	A1	19980218	EP 1995-918935	19950428
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE				
JP 11505107	T	19990518	JP 1995-532463	19950428
PRIORITY APPLN. INFO.:			WO 1995-US5500	W 19950428

AB Antibodies with fully human variable regions against a specific antigen can be prepared by administering the antigen to a transgenic animal which has been modified to produce such antibodies in response to antigenic challenge, but whose endogenous loci have been disabled. Various subsequent manipulations can be performed to obtain either antibodies per se or analogs thereof.

L6 ANSWER 99 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 96228211 EMBASE  
 DOCUMENT NUMBER: 1996228211  
 TITLE: Biotec finds a growth industry.  
 AUTHOR: Roush W.  
 SOURCE: Science, (1996) Vol. 273, No. 5273, pp. 300-301. .  
 ISSN: 0036-8075 CODEN: SCIEAS  
 COUNTRY: United States  
 DOCUMENT TYPE: Journal; Note  
 FILE SEGMENT: 027 Biophysics, Bioengineering and Medical Instrumentation  
 030 Pharmacology  
 037 Drug Literature Index  
 LANGUAGE: English  
 ENTRY DATE: Entered STN: 28 Oct 1996  
 Last Updated on STN: 28 Oct 1996

DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

=> dis ibib abs 16 80-89

L6 ANSWER 80 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2000337455 EMBASE  
 TITLE: Treatment of leukemia, lymphoma and cancer - 13th International Symposium: Molecular Biology of Hematopoiesis: 14-18 July 2000, New York, NY, USA.  
 AUTHOR: Rose-John S.  
 CORPORATE SOURCE: S. Rose-John, Department of Biochemistry, Christian-Albrechts Universitat Kiel, Olshausenstrasse 40, D-24098 Kiel, Germany. rosejohn@biochem.uni-kiel.de  
 SOURCE: Current Opinion in Oncologic, Endocrine and Metabolic Investigational Drugs, (2000) Vol. 2, No. 4, pp. 423-425. .  
 ISSN: 1464-8466 CODEN: COODF2

COUNTRY: United Kingdom  
DOCUMENT TYPE: Journal; Conference Article  
FILE SEGMENT: 037 Drug Literature Index  
016 Cancer  
025 Hematology  
022 Human Genetics  
030 Pharmacology  
026 Immunology, Serology and Transplantation  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
ENTRY DATE: Entered STN: 13 Oct 2000  
Last Updated on STN: 13 Oct 2000

AB This conference included sessions covering the clinical aspects of methodological CD34+ cell expansion, identification of the true hematopoietic stem cells, cancer and coagulation therapies, pathology of anemia in cancer patients following chemotherapy, molecular biology of erythropoietin signaling, and the role of hypoxia as a regulator of malignant cell growth. Sessions on lymphopoiesis, dendritic cells and the control of the immune response were presented in addition to updates on the JAK/STAT signaling pathways of cytokine receptors and on the construction and use of novel designer cytokines in the expansion of hematopoietic progenitor cells and gene therapy of human cancer. Innovation and new strategies for bone marrow transplantation and organ transplants were discussed, and the use of p53 gene transfer in the induction of apoptosis in malignant cells was evaluated. More than 400 scientific contributions were presented as plenary talks, short communications and poster presentations; approximately 250 to 300 people attended.

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ACCESSION NUMBER: 2000178806 EMBASE  
TITLE: Applications of developmental biology to medicine and animal agriculture.  
AUTHOR: Smith R.C.; Rhodes S.J.  
CORPORATE SOURCE: Dr. R.C. Smith, Department of Biology, IUPUI, 723 W. Michigan Street, Indianapolis, IN 46202-5132, United States  
SOURCE: Progress in Drug Research, (2000) Vol. 54, pp. 213-256. .  
Refs: 221  
ISSN: 0071-786X CODEN: FAZMAE  
COUNTRY: Switzerland  
DOCUMENT TYPE: Journal; General Review  
FILE SEGMENT: 021 Developmental Biology and Teratology  
022 Human Genetics  
030 Pharmacology  
037 Drug Literature Index  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
ENTRY DATE: Entered STN: 8 Jun 2000  
Last Updated on STN: 8 Jun 2000

AB With the complete sequence of the human genome expected by winter 2001, genomic-based drug discovery efforts of the pharmaceutical industry are focusing on finding the relatively few therapeutically useful genes from among the total gene set. Methods to rapidly elucidate gene function will have increasing value in these investigations. The use of model organisms in functional genomics has begun to be recognized and exploited and is one example of the emerging use of the tools of developmental biology in recent drug discovery efforts. The use of protein products expressed during embryogenesis and the use of certain pluripotent cell populations (stem cells) as candidate therapeutics are other applications of developmental biology to the treatment of human diseases. These agents may be used to repair damaged or diseased tissues by inducing or directing developmental programs that recapitulate embryonic processes to replace specialized cells. The activation or silencing of embryonic genes in the

disease state, particularly those encoding transcription factors, is another avenue of exploitation. Finally, the direct drug-induced manipulation of embryonic development is a unique application of developmental biology in animal agriculture.

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ACCESSION NUMBER: 2000120188 EMBASE  
TITLE: Prognostic significance of anemia and role of erythropoietin in radiation therapy.  
AUTHOR: Smaniotto D.; Luzi S.; Morganti A.G.; Cellini N.  
CORPORATE SOURCE: Dr. D. Smaniotto, Istituto di Radiologia, Universita Cattolica del Sacro Cuore, Policlinico A. Gemelli, largo A. Gemelli 8, 00168 Roma, Italy. radioterapia2.3e@rm.unicatt.it  
SOURCE: Tumori, (2000) Vol. 86, No. 1, pp. 17-23. .  
Refs: 36  
ISSN: 0300-8916 CODEN: TUMOAB  
COUNTRY: Italy  
DOCUMENT TYPE: Journal; General Review  
FILE SEGMENT: 014 Radiology  
016 Cancer  
037 Drug Literature Index  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
ENTRY DATE: Entered STN: 13 Apr 2000  
Last Updated on STN: 13 Apr 2000

AB Anemia represents a common finding in cancer patients, especially at an advanced stage. Anemia has an impact on the quality of life and at the same time seems to markedly limit the disease control that can be achieved with radiotherapy. The results of a series of clinical studies published in the last decade allow some general observations: 1. the administration of erythropoietin, especially if associated to ferrous sulfate is able to increase hemoglobin levels in cancer patients undergoing radiation therapy (combined with concomitant chemotherapy); 2. erythropoietin stimulation of hemoglobin in anemia decreases the need for blood transfusion in cancer patients; 3. tumor response to radiation therapy appears to be enhanced by erythropoietin-induced hemoglobin increase. Further clinical studies are required for assessment of indications, identification of optimal administration modalities, cost-analysis of this promising therapy for patients undergoing radiation therapy.

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ACCESSION NUMBER: 2000081357 EMBASE  
TITLE: Manifestations and treatment of Schimke immuno-osseous dysplasia: 14 new cases and a review of the literature.  
AUTHOR: Boerkoel C.F.; O'Neill S.; Andre J.L.; Benke P.J.; Bogdanovic R.; Bulla M.; Burguet A.; Cockfield S.; Cordeiro I.; Ehrich J.H.H.; Frund S.; Geary D.F.; Ieshima A.; Illies F.; Joseph M.W.; Kaitila I.; Lama G.; Leheup B.; Ludman M.D.; McLeod D.R.; Medeira A.; Milford D.V.; Ormala T.; Rener-Primec Z.; Santava A.; Santos H.G.; Schmidt B.; Smith G.C.; Spranger J.; Zupancic N.; Weksberg R.  
CORPORATE SOURCE: R. Weksberg, Hospital for Sick Children, Div. of Clinic./Metabolic Genetics, University of Toronto, 555 University Avenue, Toronto, Ont. M5G 1X8, Canada. rweksbg@sickkids.on.ca  
SOURCE: European Journal of Pediatrics, (2000) Vol. 159, No. 1-2, pp. 1-7. .  
Refs: 23  
ISSN: 0340-6199 CODEN: EJPEDT  
COUNTRY: Germany

DOCUMENT TYPE: Journal; General Review  
 FILE SEGMENT: 007 Pediatrics and Pediatric Surgery  
 022 Human Genetics  
 037 Drug Literature Index  
 LANGUAGE: English  
 SUMMARY LANGUAGE: English  
 ENTRY DATE: Entered STN: 16 Mar 2000  
 Last Updated on STN: 16 Mar 2000

AB Schimke immuno-osseous dysplasia (SIOD) is a rare autosomal recessive spondylo-epiphyseal dysplasia. The characteristic features of SIOD include 1) short stature with hyperpigmented macules and an unusual facies, 2) proteinuria with progressive renal failure, 3) lymphopenia with recurrent infections, and 4) cerebral ischaemia. Although 25 patients have been reported with this disorder, the clinical course and phenotype of SIOD are not well characterized. This report summarizes the clinical findings, course and treatment of reported patients and includes 14 additional patients with SIOD. We emphasize the high incidence of cerebral ischaemia and ocular abnormalities, define the high incidence of thyroid dysfunction and blood cytopenia, and confirm the absence of effective and durable medical therapies. Conclusion: Schimke immuno-osseous dysplasia is a multi-system autosomal recessive disorder with variable expression that affects the skeletal, renal, immune, vascular, and haematopoietic systems. Medical therapy is limited especially for more severely affected individuals.

L6 ANSWER 84 OF 113 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1999:795994 CAPLUS  
 DOCUMENT NUMBER: 132:31744  
 TITLE: Gene probes used for genetic profiling in healthcare screening and planning  
 INVENTOR(S): Roberts, Gareth Wyn  
 PATENT ASSIGNEE(S): Genostic Pharma Ltd., UK  
 SOURCE: PCT Int. Appl., 745 pp.  
 CODEN: PIXXD2  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 2  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9964627	A2	19991216	WO 1999-GB1780	19990604
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
PRIORITY APPLN. INFO.:				
			GB 1998-12099	A 19980606
			GB 1998-13291	A 19980620
			GB 1998-13611	A 19980624
			GB 1998-13835	A 19980627
			GB 1998-14110	A 19980701
			GB 1998-14580	A 19980707
			GB 1998-15438	A 19980716
			GB 1998-15574	A 19980718
			GB 1998-15576	A 19980718
			GB 1998-16085	A 19980724
			GB 1998-16086	A 19980724
			GB 1998-16921	A 19980805
			GB 1998-17097	A 19980807

GB 1998-17200 A 19980808  
 GB 1998-17632 A 19980814  
 GB 1998-17943 A 19980819

AB There is considerable evidence that significant factor underlying the individual variability in response to disease, therapy and prognosis lies in a person's genetic make-up. There have been numerous examples relating that polymorphisms within a given gene can alter the functionality of the protein encoded by that gene thus leading to a variable physiol. response. In order to bring about the integration of genomics into medical practice and enable design and building of a technol. platform which will enable the everyday practice of mol. medicine a way must be invented for the DNA sequence data to be aligned with the identification of genes central to the induction, development, progression and outcome of disease or physiol. states of interest. According to the invention, the number of genes and their configurations (mutations and polymorphisms) needed to be identified in order to provide critical clin. information concerning individual prognosis is considerably less than the 100,000 thought to comprise the human genome. The identification of the identity of the core group of genes enables the invention of a design for genetic profiling technologies which comprises of the identification of the core group of genes and their sequence variants required to provide a broad base of clin. prognostic information - "genostics". The "Genostic" profiling of patients and persons will radically enhance the ability of clinicians, healthcare professionals and other parties to plan and manage healthcare provision and the targeting of appropriate healthcare resources to those deemed most in need. The use of this invention could also lead to a host of new applications for such profiling technologies, such as identification of persons with particular work or environment related risk, selection of applicants for employment, training or specific opportunities or for the enhancing of the planning and organization of health services, education services and social services.

L6 ANSWER 85 OF 113 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1999:795993 CAPLUS  
 DOCUMENT NUMBER: 132:31743  
 TITLE: Gene probes used for genetic profiling in healthcare screening and planning  
 INVENTOR(S): Roberts, Gareth Wyn  
 PATENT ASSIGNEE(S): Genostic Pharma Limited, UK  
 SOURCE: PCT Int. Appl., 149 pp.  
 CODEN: PIXXD2  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 2  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9964626	A2	19991216	WO 1999-GB1779	19990604
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2330929	A1	19991216	CA 1999-2330929	19990604
AU 9941586	A	19991230	AU 1999-41586	19990604
AU 766544	B2	20031016		
AU 9941587	A	19991230	AU 1999-41587	19990604
GB 2339200	A	20000119	GB 1999-12914	19990604
GB 2339200	B	20010912		

EP 1084273 A1 20010321 EP 1999-925207 19990604  
 R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,  
 IE, FI  
 JP 2003528564 T 20030930 JP 2000-553616 19990604  
 US 2003198970 A1 20031023 US 2002-206568 20020729  
 PRIORITY APPLN. INFO.:

GB 1998-12098 A 19980606  
 GB 1998-28289 A 19981223  
 GB 1998-16086 A 19980724  
 GB 1998-16921 A 19980805  
 GB 1998-17097 A 19980807  
 GB 1998-17200 A 19980808  
 GB 1998-17632 A 19980814  
 GB 1998-17943 A 19980819  
 US 1999-325123 B1 19990603  
 WO 1999-GB1779 W 19990604

AB There is considerable evidence that significant factor underlying the individual variability in response to disease, therapy and prognosis lies in a person's genetic make-up. There have been numerous examples relating that polymorphisms within a given gene can alter the functionality of the protein encoded by that gene thus leading to a variable physiol. response. In order to bring about the integration of genomics into medical practice and enable design and building of a technol. platform which will enable the everyday practice of mol. medicine a way must be invented for the DNA sequence data to be aligned with the identification of genes central to the induction, development, progression and outcome of disease or physiol. states of interest. According to the invention, the number of genes and their configurations (mutations and polymorphisms) needed to be identified in order to provide critical clin. information concerning individual prognosis is considerably less than the 100,000 thought to comprise the human genome. The identification of the identity of the core group of genes enables the invention of a design for genetic profiling technologies.

L6 ANSWER 86 OF 113 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1999:594848 CAPLUS  
 DOCUMENT NUMBER: 131:223977  
 TITLE: Compositions and methods for inducing neovascularization using a vascularization modulating agent such as GM-CSF  
 INVENTOR(S): Isner, Jeffrey M.; Asahara, Takayuki  
 PATENT ASSIGNEE(S): St. Elizabeth's Medical Center, USA  
 SOURCE: PCT Int. Appl., 74 pp.  
 CODEN: PIXXD2  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 2  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9945775	A1	19990916	WO 1999-US5130	19990309
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2322559	A1	19990916	CA 1999-2322559	19990309
AU 9930737	A	19990927	AU 1999-30737	19990309
AU 766238	B2	20031009		
EP 1061800	A1	20001227	EP 1999-912344	19990309
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,				

IE, FI  
 JP 2002506008 T 20020226 JP 2000-535201 19990309  
 US 2004228835 A1 20041118 US 2003-714574 20031114  
 PRIORITY APPLN. INFO.: US 1998-77262P P 19980309  
 US 1999-265041 A3 19990309  
 WO 1999-US5130 W 19990309  
 US 2000-698323 A1 20001027

AB The present invention generally provides methods for modulating formation of new blood vessels. In one embodiment, the methods include administering to a mammal an effective amount of a vascularization modulating agent (such as granulocyte macrophage-colony stimulating factor) sufficient to form the new blood vessels. Addnl. provided are methods for preventing or reducing the severity of blood vessel damage in a mammal which methods preferably include administering to the mammal an effective amount of GM-CSF or another vascularization modulating agent. Instead of the proteins themselves being administered, the DNA encoding for the vascularization modulating agents can be administered. Addnl., the vascularization modulating agent can also be coadministered with at least one angiogenic protein. In addition to administering the vascularization modulating agent to treat ischemic tissue, it's also possible to contact isolated endothelial progenitor cells (EPCs) with an amount of an angiogenic protein sufficient to induce proliferation of the EPCs and then administer the proliferated EPCs to treat the ischemic tissue. Provided also as part of this invention are pharmaceutical products and kits for inducing formation of new blood vessels in the mammal.

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 87 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1999302671 EMBASE  
 TITLE: [Efficiency, safety and tolerability of recombinant human Interleukin-3 (rhIL-3) as supportive treatment under dose-intensified Carboplatin- chemotherapy in ovarian cancer patients with special regard to thrombocytopenia]. EFFIZIENZ, SICHERHEIT UND VERTRAGLICHKEIT VON REKOMBINANTEM HUMANEN INTERLEUKIN-3 (RHIL-3) ALS SUPPORTIVE THERAPIE BEGLEITEND ZUR DOSISINTENSIVIERTEN CARBOPLATIN-HALTIGEN CHEMOTHERAPIE BEI PATIENTINNEN MIT OVARIALKARZINOM UNTER BESONDERER BERUICKSICHTIGUNG DER THROMBOZYTOPENIE.

AUTHOR: Meden H.; Fock M.; Krauss T.; Kuhn W.  
 CORPORATE SOURCE: Dr. H. Meden, Universitäts-Frauenklinik Gottingen, Robert-Koch-Strasse 40, D-37075 Gottingen, Germany. hmeden@med.uni-goettingen.de  
 SOURCE: Zentralblatt fur Gynakologie, (1999) Vol. 121, No. 8, pp. 375-383.

Refs: 20  
 ISSN: 0044-4197 CODEN: ZEGYAX

COUNTRY: Germany  
 DOCUMENT TYPE: Journal; Article  
 FILE SEGMENT: 010 Obstetrics and Gynecology  
 016 Cancer  
 026 Immunology, Serology and Transplantation  
 037 Drug Literature Index  
 038 Adverse Reactions Titles

LANGUAGE: German  
 SUMMARY LANGUAGE: English; German  
 ENTRY DATE: Entered STN: 10 Sep 1999  
 Last Updated on STN: 10 Sep 1999

AB Objective: In a prospective, randomized, placebo-controlled double-blind trial we evaluated to what extent a dose-intensification of adjuvant chemotherapy is possible with the help of Interleukin-3 (rhIL-3).



Material and Methods: following initial surgery, 12 patients with primary ovarian cancer have been treated with Carboplatin and Cyclophosphamide (dosage: AUC 4 according to Calvert). After randomisation, a group of 6 patients prophylactically received rhIL-3 against myelosuppression on days 3-12 of the cycle, in contrast to a group of 6 patients who received placebo-injections. Results: The patients treated with rhIL-3 showed less hematologic side-effects. Adherence to 4-weekly chemotherapy courses was more frequent in the rhIL-3-group (73 % vs. 44 %,  $p = 0.005$ ). An intensification of the chemotherapy with 3-weekly courses did not succeed significantly. Observed side-effects of rhIL-3-therapy were headaches, fever, flu-like symptoms, rashes and blisters at the site of injection which excluded 2 of 6 patients from the study. Conclusions: Supportive rhIL-3 to adjuvant Carboplatin-based chemotherapy enables a better keeping of 4-weekly courses in contrast to the placebo-group due to faster recovery of hematologic parameters. Due to the side-effects, of IL-3, this cytokine cannot be recommended for routine clinical use.

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ACCESSION NUMBER: 1999248033 EMBASE  
 TITLE: Changes of cytokine mRNA in peripheral blood mononuclear cells from unresectable non-small cell lung cancer patients before and after clarithromycin therapy.  
 AUTHOR: Majima T.; Mikasa K.; Hamada K.; Konish M.; Maeda K.; Sakamoto M.; Yoshimoto E.; Murakawa K.; Ueda K.; Kita E.; Narita N.  
 CORPORATE SOURCE: T. Majima, Internal Medicine II, Nara Medical University, 840 Shijouchou, Kashihara, Nara 634, Japan  
 SOURCE: Japanese Journal of Chemotherapy, (1999) Vol. 47, No. 6, pp. 345-348. .  
 Refs: 13  
 ISSN: 1340-7007 CODEN: NKRZE5  
 COUNTRY: Japan  
 DOCUMENT TYPE: Journal; Article  
 FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis  
 016 Cancer  
 037 Drug Literature Index  
 LANGUAGE: Japanese  
 SUMMARY LANGUAGE: English; Japanese  
 ENTRY DATE: Entered STN: 2 Aug 1999  
 Last Updated on STN: 2 Aug 1999

AB We have reported that long term clarithromycin (CAM) therapy improves the survival time of patients with non-small cell lung cancer. In the present study, we examined peripheral blood mononuclear cells for changes in cytokine mRNA by RT-PCR before and after CAM therapy. The study included 15 patients with unresectable non-small cell lung cancer. Before CAM therapy, 13 patients received basic therapy consisting of chemotherapy, radiotherapy or both. Two patients received no basic therapy. Interleukin-10 (IL-10), Interleukin-12 (IL-12), Interferon-gamma (IFN- $\gamma$ ) mRNA were measured before and at one and three months after starting CAM therapy. IL-12 and IFN- $\gamma$  mRNA were significantly increased, and IL-10 mRNA was decreased. The results suggest that CAM exhibits an antitumor effect that promotes Th-lymphocytes shows a Th 1-like cytokine production.

L6 ANSWER 89 OF 113 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2000:177684 CAPLUS .  
 DOCUMENT NUMBER: 132:292537  
 TITLE: Human cytokines modulate arterial vascular tone via endothelial receptors  
 AUTHOR(S): Iversen, Per Ole; Nicolaysen, Anne; Kvernebo, Knut; Benestad, Haakon B.; Nicolaysen, Gunnar  
 CORPORATE SOURCE: Department of Physiology, Institute of Basic Medical Sciences, University of Oslo, Oslo, N-0317, Norway

SOURCE: Pfluegers Archiv (1999), 439(1-2), 93-100  
CODEN: PFLABK; ISSN: 0031-6768  
PUBLISHER: Springer-Verlag  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB Only a few cytokines have been tested for their possible role in modulating vascular function. Moreover, no direct effect of cytokines on vascular tone has yet been thoroughly studied. We therefore examined whether a wide range of well-defined cytokines could directly affect vascular tone in isolated human arterial and venous segments from various organs. We found that the cytokines stem cell factor (maximal response with 1 mM), granulocyte colony-stimulating factor (0.1 mM) and erythropoietin (1 mM) relaxed, while tumor necrosis factor  $\alpha$  (0.1 mM), interleukin (IL) 6 (10 mM) and IL-10 (0.1 mM) induced contraction of arterial but not of venous segments. The cytokines (maximal concentration tested was 1 mM) IL-3, IL-5, IL-13, macrophage colony-stimulating factor and granulocyte-macrophage colony-stimulating factor had no apparent effects on either arterial or venous tone. These vascular effects were endothelium-dependent as denuded arteries did not respond to any cytokine, and inhibition of nitric oxide synthase or endothelin receptor A abrogated the cytokine-induced changes in vascular tone. With immunohistochem. we found receptors for the active cytokines on the arterial endothelium. In conclusion, several cytokines may modulate arterial vascular tone via endothelium-dependent mechanisms. Therefore cytokines might significantly modify blood supply to inflamed or ischemic tissues with elevated local concns. of cytokines.

REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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ENTRY	SESSION
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LAST RELOADED: Jan 5, 2007 (20070105/UP).

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(Y)/N:y

L6 ANSWER 70 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2003463279 EMBASE  
TITLE: Treatment of hepatitis C virus infection in the allograft.  
AUTHOR: Neuberger J.  
CORPORATE SOURCE: J. Neuberger, Liver Unit, Queen Elizabeth Hospital,  
Birmingham B15 2TH, United Kingdom..  
James.Neuberger@uhb.nhs.uk  
SOURCE: Liver Transplantation, (2003) Vol. 9, No. 11, pp.  
S101-S108.

Refs: 46  
 ISSN: 1527-6465 CODEN: LITRFO  
 COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article  
 FILE SEGMENT: 009 Surgery  
 030 Pharmacology  
 036 Health Policy, Economics and Management  
 037 Drug Literature Index  
 038 Adverse Reactions Titles  
 048 Gastroenterology  
 LANGUAGE: English  
 SUMMARY LANGUAGE: English  
 ENTRY DATE: Entered STN: 1 Dec 2003  
 Last Updated on STN: 1 Dec 2003

AB Key Points 1. Recurrence of hepatitis C virus (HCV) in the graft is associated with a reduced quality of life and worse graft survival. 2. Pretransplantation, the severity of HCV recurrence may be reduced by reducing the pretransplantation load, by avoiding the use of organs from older donors, and by reducing the ischemic times. The effect of split livers on recurrence rates is uncertain. 3. The optimal immunosuppression regime has not been established but a heavy induction regime and treatment for acute rejection are associated with more viral replication and more graft damage. 4. Presently, there is no convincing evidence for preemptive treatment of HCV. 5. There are many studies on the effect of interferon with and without ribavirin for the treatment of HCV hepatitis. However, few are prospective, randomized, and controlled. 6. The current best treatment is with pegylated interferon and ribavirin; the dose and duration of treatment need to be established. Side-effects of treatment are common and reduction/withdrawal is frequent, but the regime is cost-effective. 7. The role of newer treatments remains to be established.

L6 ANSWER 71 OF 113 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2002:184917 CAPLUS

DOCUMENT NUMBER: 136:268103

TITLE: Method for modifying synthetic proteins with branched water-soluble polymer to improve their biol. activity or pharmacokinetic properties

INVENTOR(S): Kochendoerfer, Gerd; Kent, Stephen B. H.; Botti, Paolo; Low, Donald W.; Bradburne, James A.; Chen, Shiah-Yun; Cressman, Sonya; Hunter, Christie L.; Kent, Stephen B. H.; Low, Donald W.; Wilken, Jill G.

PATENT ASSIGNEE(S): Gryphon Sciences, USA

SOURCE: PCT Int. Appl., 245 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 4

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002020033	A1	20020314	WO 2001-US21930	20010712
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2412278	A1	20020314	CA 2001-2412278	20010712
AU 2001073385	A5	20020322	AU 2001-73385	20010712
BR 2001013579	A	20030617	BR 2001-13579	20010712

EP 1318827	A1	20030618	EP 2001-952654	20010712
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2004508338	T	20040318	JP 2002-524516	20010712
US 2004115774	A1	20040617	US 2003-332385	20030108
US 7118737	B2	20061010		
NO 2003001047	A	20030508	NO 2003-1047	20030306
US 2006233747	A1	20061019	US 2006-446419	20060605
PRIORITY APPLN. INFO.:			US 2000-231339P	P 20000908
			US 2000-236377P	P 20000929
			WO 2001-US21930	W 20010712
			US 2003-332385	A1 20030108

AB The present invention relates to methods and compns. for modifying peptides, polypeptides and proteins with polymers, especially glyco-mimetic polymers, so as to improve their biol. activity or pharmacokinetic properties. The invention provides seven synthetic erythropoiesis stimulating proteins and four RANTES derivs. having one or more branched water-soluble polymers attached thereto. The invention further provides methods and uses for such polymer-modified peptides, polypeptides and proteins. The invention is particularly suitable for use in the synthesis of polymer-modified synthetic bioactive proteins (Figure 1D), and of pharmaceutical compns. that contain such proteins.

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 72 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 8

ACCESSION NUMBER: 2002415044 EMBASE  
 TITLE: Neurite outgrowth promoting effect of cytokines on cultured rat embryonic ventral spinal cord neurons.  
 AUTHOR: Ichikawa Y.  
 CORPORATE SOURCE: Y. Ichikawa, 4th Department of Internal Medicine, Toho University School of Medicine, Chiba-ken, Japan  
 SOURCE: Journal of the Medical Society of Toho University, (2002) Vol. 49, No. 4-5, pp. 258-264. .  
 Refs: 25  
 ISSN: 0040-8670 CODEN: TOIZAG  
 COUNTRY: Japan  
 DOCUMENT TYPE: Journal; Article  
 FILE SEGMENT: 008 Neurology and Neurosurgery  
 026 Immunology, Serology and Transplantation  
 LANGUAGE: Japanese  
 SUMMARY LANGUAGE: English; Japanese  
 ENTRY DATE: Entered STN: 2 Dec 2002  
 Last Updated on STN: 2 Dec 2002

AB Background: To clarify the possible neurite outgrowth promoting effect (NPE) of cytokines including interleukin-3 (IL-3), IL-6, erythropoietin (EPO), granulocyte-colony stimulating factor (G-CSF) and tumor necrosis factor- $\beta$  (TNF- $\beta$ ) on cultured ventral spinal cord neurons of rat embryos, I studied the NPE effect of these cytokines on the primary explant cultures of ventral spinal cord neurons of fetal rats. Methods: The spinal cords from 13 or 14 embryonic day rat embryos were explanted. Explants in the culture medium were added cytokines at different concentrations, given on the first day of culture in the form of a single administration. For quantitative analysis of the NPE of these cytokines, neurite length was directly measured at the 7th culture day. Results: Neurite length was significantly increased in IL-3, IL-6 and EPO treated cultures and their effects seemed to be concentration-dependent in their effective concentration ranges. However, G-CSF and TNF- $\beta$  had no neurite elongation effects at any concentration. Conclusion: These results suggest that several cytokines may have therapeutic potential in damaged motor neuron disorders, such as amyotrophic lateral sclerosis.

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ACCESSION NUMBER: 2002323900 EMBASE  
TITLE: Cytokines in coagulation and thrombosis: A preclinical and clinical review.  
AUTHOR: Joseph L.; Fink L.M.; Hauer-Jensen M.  
CORPORATE SOURCE: Dr. L. Joseph, Department of Pathology, Univ. of Arkansas for Med. Sciences, 4301 West Markham, Little Rock, AR 72205, United States. josephlija@uams.edu  
SOURCE: Blood Coagulation and Fibrinolysis, (2002) Vol. 13, No. 2, pp. 105-116. .  
Refs: 103  
ISSN: 0957-5235 CODEN: BLFIE7  
COUNTRY: United Kingdom  
DOCUMENT TYPE: Journal; Article  
FILE SEGMENT: 005 General Pathology and Pathological Anatomy  
025 Hematology  
026 Immunology, Serology and Transplantation  
037 Drug Literature Index  
038 Adverse Reactions Titles  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
ENTRY DATE: Entered STN: 26 Sep 2002  
Last Updated on STN: 26 Sep 2002

AB The cytokine network is a complex and dynamic system, involved in numerous biological responses in the human body. This review of the current literature describes the role of cytokines and their interaction with the coagulation system, specifically in the maintenance of the thrombo-hemorrhagic balance in vivo in human subjects and in animals. In general, cytokines are thrombogenic, but they are amenable to therapeutic manipulations and hence are a potentially attractive tool in the clinician's armamentarium. Studies of the effects of cytokines in vivo are difficult because cytokines act in a very finite microenvironment and, although their actions are significant, they are transient. Most of the available clinical data related to interactions between cytokines and the coagulation system focuses on the role of tumor necrosis factor-alpha and interleukin-1 in septicemia and septic shock. However, several other cytokines and related proteins, such as platelet activating factor and plasminogen activator inhibitor, are also known to influence coagulation and thrombosis. These factors interact closely with cytokines, and have been included in this review for a better understanding of their interactions with traditional cytokines. Studies that utilize cell culture systems do not accurately model the in vivo status of this complex system and, hence, this review has excluded such studies. The role of the cytokine network in coronary artery disease, angiogenesis, or neoplasia has been addressed elsewhere by other workers and is not discussed here. By emphasizing important in vivo interactions, the intention of this review is to serve as an impetus to further translational research, both clinical and in the laboratory. .COPYRGT. 2002 Lippincott Williams & Wilkins.

L6 ANSWER 74 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2002345430 EMBASE  
TITLE: [Embryonal stem cells].  
EMBRYONALE STAMMZELLEN.  
AUTHOR: Wurm T.  
CORPORATE SOURCE: Dr. T. Wurm, Senefelderstrasse 38, 94036 Passau, Germany  
SOURCE: Deutsche Apotheker Zeitung, (5 Sep 2002) Vol. 142, No. 36, pp. 52-61. .  
Refs: 11  
ISSN: 0011-9857 CODEN: DAZE2  
COUNTRY: Germany

DOCUMENT TYPE: Journal; General Review  
FILE SEGMENT: 006 Internal Medicine  
008 Neurology and Neurosurgery  
021 Developmental Biology and Teratology  
037 Drug Literature Index  
049 Forensic Science Abstracts

LANGUAGE: German

ENTRY DATE: Entered STN: 17 Oct 2002  
Last Updated on STN: 17 Oct 2002

DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L6 ANSWER 75 OF 113 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on  
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ACCESSION NUMBER: 2003:356961 BIOSIS

DOCUMENT NUMBER: PREV200300356961

TITLE: Defective Yolk Sac Erythropoiesis in HIF-1a-Null Mice: A  
Role of Iron.

AUTHOR(S): Pastore, Yves D. [Reprint Author]; Divoky, Vladimir  
[Reprint Author]; Liu, Enli [Reprint Author]; Ponka,  
Premysl [Reprint Author]; Semenza, Gregg L. [Reprint  
Author]; Prchal, Josef T. [Reprint Author]

CORPORATE SOURCE: Medicine and Pediatric Hematology/Oncology, Baylor College  
of Medicine, Houston, TX, USA

SOURCE: Blood, (November 16 2002) Vol. 100, No. 11, pp. Abstract  
No. 2827. print.  
Meeting Info.: 44th Annual Meeting of the American Society  
of Hematology. Philadelphia, PA, USA. December 06-10, 2002.  
American Society of Hematology.  
CODEN: BLOOAW. ISSN: 0006-4971.

DOCUMENT TYPE: Conference; (Meeting)  
Conference; (Meeting Poster)  
Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 6 Aug 2003  
Last Updated on STN: 6 Aug 2003

AB Hypoxia-inducible factor-1 (HIF-1) regulates the expression of  
an array of genes including erythropoietin (EPO), vascular  
endothelial growth factor (VEGF), transferrin and its receptor (TfR);  
however, its role in the erythropoiesis remains to be defined. In  
Hifla-/- mice, targeted disruption of the Hifla gene encoding the  
O2-regulated HIF-1a subunit causes cardiac malformations, vascular  
regression, and death by embryonic day (ED) 10.5. In order to understand  
the role of HIF-1a in erythropoiesis, we studied the yolk sac (YS)  
erythroid progenitors from Hifla-/-, Hifla+/-, and wild-type (WT)  
littermate embryos. Hematopoietic progenitors from isolated YS at ED  
9-9.5 were analyzed by in vitro culture in presence of interleukin-3  
(IL-3), IL-6, Epo, stem cell factor and granulocyte-  
macrophage colony stimulating factor  
(GM-CSF). The numbers and the size of the erythroid colonies (CFU-E and  
BFU-E) from the YS of Hifla-/- embryos were decreased, and had a marked  
defect of hemoglobinization compared to erythroid colonies from WT and  
Hifla+/- YS. Neither VEGF nor high levels of Epo added to the cultures  
fully rescued these defects. Some of the differences might be related to  
a developmental arrest in the mutant embryos, as the number of somites in  
Hifla-/- embryos was significantly lower compared to the Hifla+/- and WT  
embryos (12, 20, 22 respectively). However, we hypothesized that the  
defective hemoglobinization may be due to abnormalities in iron  
metabolism, e.g. down-regulation of TfR. We cultured YS cells in the  
presence of salicylaldehyde isonicotinoyl hydrazone saturated with iron  
(Fe-SIH) which transports iron into cells independently of the TfR. In  
the presence of 100 micromolar Fe-SIH, we observed a significant increase  
in the numbers of erythroid colonies derived from YS of WT and, to a  
lesser degree, Hifla+/- embryos. Although there was no increase in the  
number of colonies from the YS of Hifla-/- embryos, the size of the

erythroid colonies and the degree of hemoglobinization was markedly improved. These results demonstrate defects in YS erythroid colony formation and hemoglobinization in Hifla-/- embryos and suggest that the latter may be associated with a disturbance of iron metabolism.

L6 ANSWER 76 OF 113 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2001:338762 CAPLUS  
DOCUMENT NUMBER: 134:362292  
TITLE: Methods of determining individual hypersensitivity to a pharmaceutical agent from gene expression profile  
INVENTOR(S): Farr, Spencer  
PATENT ASSIGNEE(S): Phase-1 Molecular Toxicology, USA  
SOURCE: PCT Int. Appl., 222 pp.  
CODEN: PIXXD2  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 1  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001032928	A2	20010510	WO 2000-US30474	20001103
WO 2001032928	A3	20020725		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM  
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-165398P P 19991105  
US 2000-196571P P 20000411

AB. The invention discloses methods, gene databases, gene arrays, protein arrays, and devices that may be used to determine the hypersensitivity of individuals to a given agent, such as drug or other chemical, in order to prevent toxic side effects. In one embodiment, methods of identifying hypersensitivity in a subject by obtaining a gene expression profile of multiple genes associated with hypersensitivity of the subject suspected to be hypersensitive, and identifying in the gene expression profile of the subject a pattern of gene expression of the genes associated with hypersensitivity are disclosed. The gene expression profile of the subject may be compared with the gene expression profile of a normal individual and a hypersensitive individual. The gene expression profile of the subject that is obtained may comprise a profile of levels of mRNA or cDNA. The gene expression profile may be obtained by using an array of nucleic acid probes for the plurality of genes associated with hypersensitivity. The expression of the genes predetd. to be associated with hypersensitivity is directly related to prevention or repair of toxic damage at the tissue, organ or system level. Gene databases arrays and apparatus useful for identifying hypersensitivity in a subject are also disclosed.

L6 ANSWER 77 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2002351769 EMBASE  
TITLE: Pharmacologically regulated cell therapy.  
AUTHOR: Neff T.; Blau C.A.  
CORPORATE SOURCE: C.A. Blau, Mailstop 357710, Health Sciences Building, University of Washington, Seattle, WA 98195, United States. tblau@u.washington.edu  
SOURCE: Blood, (1 May 2001) Vol. 97, No. 9, pp. 2535-2540. .  
Refs: 67

ISSN: 0006-4971 CODEN: BLOOAW  
COUNTRY: United States  
DOCUMENT TYPE: Journal; General Review  
FILE SEGMENT: 022 Human Genetics  
025 Hematology  
026 Immunology, Serology and Transplantation  
037 Drug Literature Index  
038 Adverse Reactions Titles  
LANGUAGE: English  
ENTRY DATE: Entered STN: 31 Oct 2002  
Last Updated on STN: 31 Oct 2002  
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L6 ANSWER 78 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2001018704 EMBASE  
TITLE: Paying for the greying of society.  
AUTHOR: Stokes R.  
SOURCE: Pharmaceutical Technology Europe, (2001) Vol. 13, No. 1, pp. 58+60.  
ISSN: 0164-6826 CODEN: PTEUFB  
COUNTRY: United Kingdom  
DOCUMENT TYPE: Journal; (Short Survey)  
FILE SEGMENT: 020 Gerontology and Geriatrics  
036 Health Policy, Economics and Management  
018 Cardiovascular Diseases and Cardiovascular Surgery  
028 Urology and Nephrology  
003 Endocrinology  
037 Drug Literature Index  
008 Neurology and Neurosurgery  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
ENTRY DATE: Entered STN: 25 Jan 2001  
Last Updated on STN: 25 Jan 2001

AB A recent US report has highlighted how all Western governments may need to work with insurers to make biotech-based drugs affordable for an ageing population.

L6 ANSWER 79 OF 113 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2000305414 EMBASE  
TITLE: The use of erythropoietin in neonates.  
AUTHOR: Ohls R.K.  
CORPORATE SOURCE: Dr. R.K. Ohls, Department of Pediatrics, ACC-3W, Univ. of New Mexico Hlth. Sci. Ctr., Albuquerque, NM 87131-5311, United States. rohls@unm.edu  
SOURCE: Clinics in Perinatology, (2000) Vol. 27, No. 3, pp. 681-696.  
Refs: 80  
ISSN: 0095-5108 CODEN: CLPEDL  
COUNTRY: United States  
DOCUMENT TYPE: Journal; General Review  
FILE SEGMENT: 007 Pediatrics and Pediatric Surgery  
025 Hematology  
036 Health Policy, Economics and Management  
037 Drug Literature Index  
038 Adverse Reactions Titles  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
ENTRY DATE: Entered STN: 14 Sep 2000  
Last Updated on STN: 14 Sep 2000

AB Although much information has been accumulated about the clinical use of Epo in preterm infants, many questions remain unanswered. The evolution of clinical practice in the care of extremely ill, preterm infants



continues to affect the number of transfusions required during hospitalization. Decreasing phlebotomy losses and instituting standardized transfusion guidelines have both been shown significantly to decrease the transfusion requirements of preterm infants. The administration of Epo likely decreases transfusion need even further; however, the direct impact of each of these actions has not been studied prospectively. It is likely that the combination of instituting rigorous and standardized transfusion guidelines, decreasing phlebotomy losses, and the appropriate use of Epo will have the greatest impact in decreasing transfusion requirements in all preterm and term neonates, regardless of the cause of their anemia.

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NEWS	11	DEC 11	CAS REGISTRY chemical nomenclature enhanced
NEWS	12	DEC 14	WPIDS/WPINDEX/WPIX manual codes updated
NEWS	13	DEC 14	GBFULL and FRFULL enhanced with IPC 8 features and functionality
NEWS	14	DEC 18	CA/CAPLUS pre-1967 chemical substance index entries enhanced with preparation role
NEWS	15	DEC 18	CA/CAPLUS patent kind codes updated
NEWS	16	DEC 18	MARPAT to CA/CAPLUS accession number crossover limit increased to 50,000
NEWS	17	DEC 18	MEDLINE updated in preparation for 2007 reload
NEWS	18	DEC 27	CA/CAPLUS enhanced with more pre-1907 records
NEWS	19	JAN 08	CHEMLIST enhanced with New Zealand Inventory of Chemicals
NEWS EXPRESS		NOVEMBER 10	CURRENT WINDOWS VERSION IS V8.01c, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 25 SEPTEMBER 2006.
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granulocyte(w)colony(w)stimulating(w)factor)

L1 69446 (GMCSF OR GRANULOCYTE(W) MACROPHAGE(W) COLONY(W) STIMULATING(W)  
FACTOR OR GCSF GRANULOCYTE(W) COLONY(W) STIMULATING(W) FACTOR)

=> s l1 and (ischemia or hypoxia or stroke or neurodegenerative or neurological)

L2 672 L1 AND (ISCHEMIA OR HYPOXIA OR STROKE OR NEURODEGENERATIVE OR  
NEUROLOGICAL)

=> s l2 and treatment

L3 240 L2 AND TREATMENT

=> s l3 and (interleukin or erythropoietin)

L4 23 L3 AND (INTERLEUKIN OR ERYTHROPOIETIN)

=> dup rem l4

PROCESSING COMPLETED FOR L4

L5 23 DUP REM L4 (0 DUPLICATES REMOVED)

=> dis his

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FILE 'MEDLINE, EMBASE, BIOSIS, CAPLUS' ENTERED AT 10:58:48 ON 11 JAN 2007

L1 69446 S (GMCSF OR GRANULOCYTE(W)MACROPHAGE(W)COLONY(W)STIMULATING(W)F

L2 672 S L1 AND (ISCHEMIA OR HYPOXIA OR STROKE OR NEURODEGENERATIVE OR

L3 240 S L2 AND TREATMENT

L4 23 S L3 AND (INTERLEUKIN OR ERYTHROPOIETIN)

L5 23 DUP REM L4 (0 DUPLICATES REMOVED)

=> dis ibib abs l5 15-23

L5 ANSWER 15 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2003:282718 CAPLUS

DOCUMENT NUMBER: 138:282352

TITLE: Traversal of nucleic acid molecules through a tissue  
fluid space and expression in repair cells

INVENTOR(S): Sosnowski, Barbara A.; Pierce, Glenn

PATENT ASSIGNEE(S): Selective Genetics, Inc., USA

SOURCE: PCT Int. Appl., 95 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003029429	A2	20030410	WO 2002-US31546	20021002
WO 2003029429	A3	20040401		
WO 2003029429	A9	20040701		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2002343475	A1	20030414	AU 2002-343475	20021002
US 2003148979	A1	20030807	US 2002-264284	20021002
EP 1438413	A2	20040721	EP 2002-780419	20021002

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK

PRIORITY APPLN. INFO.: US 2001-327513P P 20011003  
WO 2002-US31546 W 20021002

AB Disclosed are methods for use in transferring nucleic acids into cells at a wound site associated with a fluid space. These gene transfer protocols are suitable for use in transferring various nucleic acids into cartilage, cardiac muscle, and other tissues, and have many uses including treating diseases such as arthritis and ischemic heart disease, and promoting wound healing. The invention further disclosed pharmaceutical compns. that may be used in the practice of the invention to transfer the nucleic acid of interest. Such compns. include any multi-partitioned biocompatible matrix in combination with multiple nucleic acids of interest. Thus, collagen collagen-immobilized fibroblast growth factor (FGF) genes induce angiogenesis in vitro, and FGF gene delivery to skeletal muscle wounds induces both angiogenesis and arteriogenesis and well as induces myocyte regeneration.

L5 ANSWER 16 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2003:678274 CAPLUS

DOCUMENT NUMBER: 139:193995

TITLE: Pluripotent embryonic-like stem cells from adult tissues and their culture and therapeutic uses

INVENTOR(S): Young, Henry E.; Lucas, Paul A.

PATENT ASSIGNEE(S): USA

SOURCE: U.S. Pat. Appl. Publ., 186 pp.

CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003161817	A1	20030828	US 2001-820320	20010328
US 2005255588	A1	20051117	US 2005-29763	20050105

PRIORITY APPLN. INFO.: US 2001-820320 B1 20010328

AB Pluripotent stem cells with properties similar to pluripotent embryonic stem cells are purified from adult tissues for therapeutic and investigative use. The invention further relates to methods of purifying pluripotent embryonic-like stem cells and to compns., cultures and clones thereof. The present invention also relates to a method of transplanting the pluripotent stem cells of the present invention in a mammalian host, such as human, comprising introducing the stem cells, into the host. The invention further relates to methods of in vivo administration of a

protein or gene of interest comprising transfecting a pluripotent stem cell with a construct comprising DNA which encodes a protein of interest and then introducing the stem cell into the host where the protein or gene of interest is expressed. The present also relates to methods of producing mesodermal, endodermal or ectodermal lineage-committed cells by culturing or transplantation of the pluripotent stem cells of the present invention.

L5 ANSWER 17 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2003:1013125 CAPLUS  
DOCUMENT NUMBER: 140:65078  
TITLE: Reduced side-effect hemoglobin compositions  
INVENTOR(S): Looker, Douglas L.; Apostol, Izydor Z.; Brucker, Eric A.; Doyle, Michael P.; Foster, David L.; Glascock, Christopher B.; Hartman, James C.; Lee, Geoffrey F.; Lemon, Douglas D.; Moore, Edwin G.; Richards, Jane P.; Schick, Michael R.; Trimble, Stephen P.; Pereira, David; Hai, Ton-That; Burhop, Kenneth E.  
PATENT ASSIGNEE(S): Baxter International Inc., USA; Baxter Healthcare S.A.  
SOURCE: U.S., 62 pp., Cont.-in-part of U.S. 6,455,676.  
CODEN: USXXAM  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 3  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6670323	B1	20031230	US 2000-709914	20001110
WO 9850430	A2	19981112	WO 1998-US8861	19980501
WO 9850430	A3	19990401		

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW

RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

US 6455676	B1	20020924	US 2000-403208	20000425
US 2004259769	A1	20041223	US 2003-747580	20031229

PRIORITY APPLN. INFO.:  
WO 1998-US8861 W 19980501  
US 1999-165289P P 19991112  
US 2000-403208 A2 20000425  
US 1997-45364P P 19970502  
US 1997-57986P P 19970905  
US 2000-709914 A1 20001110

AB The invention relates to novel Hb compns., particularly novel recombinant mutant Hb compns., which eliminate or substantially reduce 1) the creation of heart lesions, 2) gastrointestinal discomfort, 3) pressor effects, and 4) endotoxin hypersensitivity associated with the administration of extracellular Hb compns. in various therapeutic applications. Applications described include treatments for anemia, head injury, hemorrhage or hypovolemia, ischemia, cachexia, sickle cell crisis and stroke; enhancing cancer treatments; stimulating hematopoiesis; improving repair of phys. damaged tissues; alleviating cardiogenic shock; and shock resuscitation.

REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 18 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2003:573223 CAPLUS  
DOCUMENT NUMBER: 139:111699  
TITLE: Uses of G-CSF, GM-CSF and SCF in conjunction with

other growth factors for the mobilization of stem cells as a new therapeutic approach to cerebrovascular and spinal cord injury therapy

INVENTOR(S): Pourquier, Didier; Moukoko, Didier  
PATENT ASSIGNEE(S): Fr.  
SOURCE: Fr. Demande, 24 pp.

CODEN: FRXXBL

DOCUMENT TYPE: Patent

LANGUAGE: French

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
FR 2834898	A1	20030725	FR 2002-610	20020118
FR 2834898	B1	20050610		
WO 2003061685	A1	20030731	WO 2003-FR13	20030106
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW			
RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
EP 1465653	A1	20041013	EP 2003-712209	20030106
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK			

PRIORITY APPLN. INFO.: FR 2002-610 A 20020118  
WO 2003-FR13 W 20030106

AB The invention relates to a new therapeutic application of at least of the factors chosen among G-CSF (granulocyte colony-stimulating factor), GM-CSF (macrophages-granulocytes colony-stimulating factor) and the SCF (stem cell factor). This factor is to be used in the preparation of a useful drug for auxiliary treatment leading to the reconstruction of nerve fibers. G-CSF, GM-CSF and SCF are particularly useful as drugs for the treatment of ischemic or hemorrhagic cerebrovascular accidents, cerebral traumas, ischemic or hemorrhagic vascular accidents of the spinal cord, and spinal cord traumas. Administration is intended in a general way, both for human and veterinary medicine.

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 19 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2001:360039 CAPLUS

DOCUMENT NUMBER: 134:371751

TITLE: Reduced side-effect hemoglobin compositions

INVENTOR(S): Looker, Douglas L.; Apostol, Izydor Z.; Brucker, Eric A.; Doyle, Michael P.; Foster, David L.; Glascock, Christopher B.; Hartman, James C.; Lee, Geoffrey F.; Lemon, Douglas D.; Moore, Edwin G.; Richards, Jane P.; Schick, Michael R.; Trimble, Stephen P.; Pereira, David; Hai, Ton-That; Burhop, Kenneth E.

PATENT ASSIGNEE(S): Baxter Biotech Technology S.A.R.L., Switz.

SOURCE: PCT Int. Appl., 146 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 3

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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WO 2001034648	A1	20010517	WO 2000-US30857	20001110
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2391226	A1	20010517	CA 2000-2391226	20001110
EP 1233986	A1	20020828	EP 2000-980318	20001110
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2003515533	T	20030507	JP 2001-537359	20001110
AU 784195	B2	20060216	AU 2001-17597	20001110
NO 2002002229	A	20020711	NO 2002-2229	20020510
ZA 2002003817	A	20030228	ZA 2002-3817	20020514
PRIORITY APPLN. INFO.:			US 1999-165289P	P 19991112
			WO 2000-US30857	W 20001110

AB The invention relates to novel Hb compns., particularly novel recombinant mutant Hb compns., which eliminate or substantially reduce 1) the creation of heart lesions, 2) gastrointestinal discomfort, 3) pressor effects, and 4) endotoxin hypersensitivity associated with the administration of extracellular Hb compns. in various therapeutic applications. Applications described include treatments for anemia, head injury, hemorrhage or hypovolemia, ischemia, cachexia, sickle cell crisis and stroke; enhancing cancer treatments; stimulating hematopoiesis; improving repair of phys. damaged tissues; alleviating cardiogenic shock; and shock resuscitation.

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 20 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2000337455 EMBASE  
 TITLE: Treatment of leukemia, lymphoma and cancer - 13th International Symposium: Molecular Biology of Hematopoiesis: 14-18 July 2000, New York, NY, USA.  
 AUTHOR: Rose-John S.  
 CORPORATE SOURCE: S. Rose-John, Department of Biochemistry, Christian-Albrechts Universitat Kiel, Olshausenstrasse 40, D-24098 Kiel, Germany. rosejohn@biochem.uni-kiel.de  
 SOURCE: Current Opinion in Oncologic, Endocrine and Metabolic Investigational Drugs, (2000) Vol. 2, No. 4, pp. 423-425. ISSN: 1464-8466 CODEN: COODF2  
 COUNTRY: United Kingdom  
 DOCUMENT TYPE: Journal; Conference Article  
 FILE SEGMENT: 037 Drug Literature Index  
 016 Cancer  
 025 Hematology  
 022 Human Genetics  
 030 Pharmacology  
 026 Immunology, Serology and Transplantation  
 LANGUAGE: English  
 SUMMARY LANGUAGE: English  
 ENTRY DATE: Entered STN: 13 Oct 2000  
 Last Updated on STN: 13 Oct 2000

AB This conference included sessions covering the clinical aspects of methodological CD34+ cell expansion, identification of the true hematopoietic stem cells, cancer and coagulation therapies, pathology of anemia in cancer patients following chemotherapy, molecular biology of erythropoietin signaling, and the role of hypoxia as a

regulator of malignant cell growth. Sessions on lymphopoiesis, dendritic cells and the control of the immune response were presented in addition to updates on the JAK/STAT signaling pathways of cytokine receptors and on the construction and use of novel designer cytokines in the expansion of hematopoietic progenitor cells and gene therapy of human cancer. Innovation and new strategies for bone marrow transplantation and organ transplants were discussed, and the use of p53 gene transfer in the induction of apoptosis in malignant cells was evaluated. More than 400 scientific contributions were presented as plenary talks, short communications and poster presentations; approximately 250 to 300 people attended.

L5 ANSWER 21 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2000081357 EMBASE  
 TITLE: Manifestations and treatment of Schimke immuno-osseous dysplasia: 14 new cases and a review of the literature.  
 AUTHOR: Boerkoel C.F.; O'Neill S.; Andre J.L.; Benke P.J.; Bogdanovic R.; Bulla M.; Burguet A.; Cockfield S.; Cordeiro I.; Ehrich J.H.H.; Frund S.; Geary D.F.; Ieshima A.; Illies F.; Joseph M.W.; Kaitila I.; Lama G.; Leheup B.; Ludman M.D.; McLeod D.R.; Medeira A.; Milford D.V.; Ormala T.; Renner-Primec Z.; Santava A.; Santos H.G.; Schmidt B.; Smith G.C.; Spranger J.; Zupancic N.; Weksberg R.  
 CORPORATE SOURCE: R. Weksberg, Hospital for Sick Children, Div. of Clinic./Metabolic Genetics, University of Toronto, 555 University Avenue, Toronto, Ont. M5G 1X8, Canada. rweksbg@sickkids.on.ca  
 SOURCE: European Journal of Pediatrics, (2000) Vol. 159, No. 1-2, pp. 1-7. .  
 Refs: 23  
 ISSN: 0340-6199 CODEN: EJPEDT  
 COUNTRY: Germany  
 DOCUMENT TYPE: Journal; General Review  
 FILE SEGMENT: 007 Pediatrics and Pediatric Surgery  
 022 Human Genetics  
 037 Drug Literature Index  
 LANGUAGE: English  
 SUMMARY LANGUAGE: English  
 ENTRY DATE: Entered STN: 16 Mar 2000  
 Last Updated on STN: 16 Mar 2000

AB Schimke immuno-osseous dysplasia (SIOD) is a rare autosomal recessive spondylo-epiphyseal dysplasia. The characteristic features of SIOD include 1) short stature with hyperpigmented macules and an unusual facies, 2) proteinuria with progressive renal failure, 3) lymphopenia with recurrent infections, and 4) cerebral ischaemia. Although 25 patients have been reported with this disorder, the clinical course and phenotype of SIOD are not well characterized. This report summarizes the clinical findings, course and treatment of reported patients and includes 14 additional patients with SIOD. We emphasize the high incidence of cerebral ischaemia and ocular abnormalities, define the high incidence of thyroid dysfunction and blood cytopenia, and confirm the absence of effective and durable medical therapies. Conclusion: Schimke immuno-osseous dysplasia is a multi-system autosomal recessive disorder with variable expression that affects the skeletal, renal, immune, vascular, and haematopoietic systems. Medical therapy is limited especially for more severely affected individuals.

L5 ANSWER 22 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN  
 ACCESSION NUMBER: 1998:323165 CAPLUS  
 DOCUMENT NUMBER: 129:8577  
 TITLE: Methods for regulating angiogenesis  
 INVENTOR(S): Isner, Jeffrey M.; Asahara, Takayuki



PATENT ASSIGNEE(S): St. Elizabeth's Medical Center of Boston, Inc., USA  
 SOURCE: PCT Int. Appl., 57 pp.  
 CODEN: PIXXD2  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 2  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9819712	A1	19980514	WO 1997-US19935	19971106
W: AU, CA, JP				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 5980887	A	19991109	US 1996-744882	19961108
CA 2271690	A1	19980514	CA 1997-2271690	19971106
AU 9852432	A	19980529	AU 1998-52432	19971106
AU 743267	B2	20020124		
EP 941125	A1	19990915	EP 1997-947319	19971106
R: DE, FR, GB, IT				
JP 2001503427	T	20010313	JP 1998-521645	19971106
EP 1618898	A2	20060125	EP 2005-17496	19971106
R: DE, FR, GB, IT				

PRIORITY APPLN. INFO.:  
 US 1996-744882 A 19961108  
 EP 1997-947319 A3 19971106  
 WO 1997-US19935 W 19971106

AB In accordance with the present invention, EC (endothelial cell) progenitors can be used in a method for regulating angiogenesis, i.e., enhancing or inhibiting blood vessel formation, in a selected patient and in some preferred embodiments for targetting specific locations. For example, the EC progenitors can be used to enhance angiogenesis or to deliver an angiogenesis modulator, e.g. anti- or pro-angiogenic agents, resp. to sites of pathol. or utilitarian angiogenesis. Addnl., in another embodiment, EC progenitors can be used to induce reendothelialization of an injured blood vessel, and thus reduce restenosis by indirectly inhibiting smooth muscle cell proliferation.

REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 23 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1998359165 EMBASE  
 TITLE: Strategies for the use of epoetin alfa in breast cancer patients.

AUTHOR: Del Mastro L.; Venturini M.

CORPORATE SOURCE: Dr. L. Del Mastro, Oncologia Medica 1, Istit. Nazionale Ricerca Cancro, L. go Rosanna Benzi 10, 16132 Genova, Italy. mventur@hp380.ist.unige.it

SOURCE: Oncologist, (1998) Vol. 3, No. 5, pp. 314-318. .  
 Refs: 27

ISSN: 1083-7159 CODEN: OCOLF6

COUNTRY: United States.

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 016 Cancer  
 025 Hematology  
 030 Pharmacology  
 037 Drug Literature Index  
 038 Adverse Reactions Titles

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 19 Nov 1998  
 Last Updated on STN: 19 Nov 1998

AB Anemia is a common complication in cancer patients undergoing chemotherapy, and its severity depends on both the type of antineoplastic drugs and the clinical status of the patient. Breast cancer patients

undergoing standard chemotherapy develop clinically significant anemia in up to 25% of cases. This percentage, moreover, increases up to 63% when more intensive chemotherapy regimens are used. The therapeutic use of erythropoietin in anemic patients, i.e., in patients with hemoglobin levels below 9-10.5 g/dl, is able to correct the anemic status in nearly 40%-80% of such patients, but it does not completely eliminate the need of blood transfusions: 20%-40% of patients need to be transfused despite the erythropoietin treatment. An alternative strategy for optimizing the erythropoietin treatment is its use in the prevention of anemia, i.e., in patients with normal hemoglobin values but at high risk of becoming anemic. In a phase III study, we evaluated the role of erythropoietin in the prevention of anemia in breast cancer patients undergoing dose-intensive chemotherapy. Clinically significant anemia occurred in 52% (95% CI = 33-69) of control patients and in no patient (95% CI = 0-14) in the erythropoietin arm ( $p = .00001$ ). After six cycles of chemotherapy the mean hemoglobin decrease was 3.05 g/dl ( $\pm 1.0$ , 95% CI = 2.6-3.5) in the control arm and 0.8 g/dl ( $\pm 1.4$ , 95% CI = 0.3-1.4) in the erythropoietin arm. Moreover, 6.4% of control patients needed blood transfusion compared to no patients in the erythropoietin arm. Erythropoietin is active in both the treatment and the prevention of anemia in cancer patients undergoing chemotherapy. Due to its high economic cost, efforts should be made to identify subsets of patients in whom the preventive use could be cost-effective. Patients undergoing chemotherapy associated with a high risk of anemia could benefit from preventive use of erythropoietin in special circumstances, such as presence of risk of myocardial or cerebral ischemia, uncommon blood group, or religious beliefs hindering blood transfusions. Moreover, anemia prevention could be considered in patients at high risk of requiring blood transfusions, such as patients with low baseline value of hemoglobin or with a hemoglobin decrease of  $\geq 2$  g/dl after the first cycle of chemotherapy.

=> dis ibib abs l5 1-14

L5 ANSWER 1 OF 23 MEDLINE on STN  
 ACCESSION NUMBER: 2006429365 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 16856086  
 TITLE: Colony stimulating factors (including erythropoietin, granulocyte colony stimulating factor and analogues) for stroke.  
 AUTHOR: Bath P M W; Sprigg N  
 CORPORATE SOURCE: University of Nottingham, Division of Stroke Medicine, South Block D Floor, Queens Medical Centre, Nottingham, Nottinghamshire, UK NG7 2UH.. philip.bath@nottingham.ac.uk  
 SOURCE: Cochrane database of systematic reviews (Online), (2006) Vol. 3, pp. CD005207. Electronic Publication: 2006-07-19. Ref: 27  
 Journal code: 100909747. E-ISSN: 1469-493X.  
 PUB. COUNTRY: England; United Kingdom  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 General Review; (REVIEW)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 200610  
 ENTRY DATE: Entered STN: 21 Jul 2006  
 Last Updated on STN: 17 Oct 2006  
 Entered Medline: 16 Oct 2006  
 AB BACKGROUND: Colony stimulating factors (CSFs), also called haematopoietic growth factors, regulate bone marrow production of circulating red and white cells, and platelets. They have been shown to be neuroprotective in experimental stroke. Some CSFs also mobilise the release of bone marrow stem cells into the circulation. OBJECTIVES: We

systematically assessed the effects of CSFs on functional outcome and haematology measures in patients with acute or subacute stroke enrolled into randomised controlled trials. SEARCH STRATEGY: We searched the Cochrane Stroke Group Trials Register (last searched February 2005), the Cochrane Central Register of Controlled Trials (CENTRAL) (The Cochrane Library Issue 3, 2005), MEDLINE (1985 to March 2006), EMBASE (1985 to November 2005), and Science Citation Index (1985 to November 2005). In an attempt to identify further published, unpublished and ongoing trials we contacted manufacturers and principal investigators of trials (last contacted 2005). We also searched reference lists of relevant articles and reviews. SELECTION CRITERIA: Unconfounded randomised controlled trials recruiting patients with acute or subacute ischaemic or haemorrhagic stroke were included. CSFs included stem cell factor (SCF), erythropoietin (EPO), granulocyte colony stimulating factor (G-CSF), granulocyte-macrophage colony stimulating factor (GM-CSF), macrophage-colony stimulating factor (M-CSF, CSF-1), and thrombopoietin (TPO), or analogues of these. The primary outcome was functional outcome (assessed as combined death or disability and dependency using scales such as the modified Rankin Scale or Barthel Index) at the end of the trial. Secondary outcomes included safety at the end of treatment (death, impairment, deterioration, extension or recurrence), death at the end of follow up, and haematology measures (blood counts at or around day seven after treatment commenced). DATA COLLECTION AND ANALYSIS: Data on measures by intention to treat (where available) were collected and analysed as dichotomous or continuous outcomes, as relevant, using random-effects models. Heterogeneity was assessed. MAIN RESULTS: No large trials were identified. EPO therapy was associated with a non-significant reduction in neurological impairment in one small trial (n = 40 participants) but had no significant effect on haematological measures. Further small trials of EPO and G-CSF are ongoing. AUTHORS' CONCLUSIONS: No large trials of EPO, G-CSF or other colony stimulating factors have been performed and it is too early to know whether CSFs improve functional outcome.

L5 ANSWER 2 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2006323695 EMBASE  
 TITLE: Hematopoietic colony stimulating factors in cardiovascular and pulmonary remodeling: Promoters or inhibitors?  
 AUTHOR: Parissis J.; Filippatos G.; Adamopoulos S.; Li X.; Kremastinos D.Th.; Uhal B.D.  
 CORPORATE SOURCE: B.D. Uhal, Department of Physiology, Michigan State University, 3185 Biomedical/Physical Sci. Bldg., East Lansing, MI 48824-3320, United States. uhal@msu.edu  
 SOURCE: Current Pharmaceutical Design, (2006) Vol. 12, No. 21, pp. 2689-2699.  
 Refs: 120  
 ISSN: 1381-6128 CODEN: CPDEFP  
 COUNTRY: Netherlands  
 DOCUMENT TYPE: Journal; General Review  
 FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis  
 018 Cardiovascular Diseases and Cardiovascular Surgery  
 025 Hematology  
 030 Pharmacology  
 037 Drug Literature Index  
 038 Adverse Reactions Titles  
 LANGUAGE: English  
 SUMMARY LANGUAGE: English  
 ENTRY DATE: Entered STN: 27 Jul 2006  
 Last Updated on STN: 27 Jul 2006

AB Hemopoietic colony stimulating factors (HCSFs) are naturally occurred substances that are released in response to infection or inflammation and regulate the proliferation and differentiation of hemopoietic progenitor

cells. Some representative members of this peptide family induce atherogenesis through the mediation of monocyte-endothelial cell adhesive interaction and promotion of angiogenesis within the atherosclerotic plaques. HCSFs, such as granulocyte-macrophage colony-stimulating factor (GM-CSF), also promote post-infarction cardiac remodeling through the enhanced activation and infiltration of monocytes into injured myocardial tissue and through altered equilibrium of collagen deposition/degradation. On the other hand, exogenous administration of granulocyte colony-stimulating factor (G-CSF) or erythropoietin (EPO) in patients with chronic ischemic disease or recent myocardial infarction have lead to beneficial arteriogenesis or myocardial cell regeneration, thus preventing adverse cardiac remodeling. While GM-CSF may hold therapeutic potential as an inhibitor of lung fibrogenesis, G-CSF appears to promote fibrosis in the lungs. The pathophysiological role of HCSFs also depends on the timing of their action on cardiovascular remodeling, as well as on the target progenitor hematopoietic cell. This article summarizes current knowledge about the clinical and therapeutic implications of these factors in chronic artery disease, post-infarction cardiac remodeling, chronic heart failure and in pulmonary fibrosis. .COPYRGT. 2006 Bentham Science Publishers Ltd.

L5 ANSWER 3 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2005:1314101 CAPLUS

DOCUMENT NUMBER: 144:68263

TITLE: Genes showing altered levels of expression in drug-resistant leukemia and their use in diagnosis and selection of drug target for therapy

INVENTOR(S): Evans, William E.; Pieters, Rob; Cheok, Meyling H.; Den Boer, Monique L.; Yang, Wenjian

PATENT ASSIGNEE(S): St. Jude Children's Research Hospital, USA; Erasmus University Medical Center Rotterdam

SOURCE: PCT Int. Appl., 124 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005118865	A2	20051215	WO 2005-US17424	20050518
WO 2005118865	A3	20060622		
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW			
RW:	BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			

PRIORITY APPLN. INFO.: US 2004-575762P P 20040528

AB The present invention encompasses methods and compns. useful in the diagnosis and treatment of drug resistant leukemia. The invention provides a number of genes that are differentially expressed between drug resistant and drug sensitive acute lymphoblastic leukemia (ALL). These genes act as biomarkers for drug resistant leukemia, and further serve as mol. targets for drugs useful in treating drug resistant leukemia. Accordingly, the invention provides methods of diagnosing drug resistant leukemia and methods of selecting a therapy for subjects affected by drug-resistant leukemia. The invention also provides methods

for screening for compds. for treating drug-resistant leukemia, and improved methods for treating drug-resistant leukemia. Compns. of the invention include arrays, computer readable media, and kits for use in the methods of the invention.

L5 ANSWER 4 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2005:572333 CAPLUS

DOCUMENT NUMBER: 143:91472

TITLE: Methods of treating neurological conditions with hematopoietic growth factors

INVENTOR(S): Schaebitz, Wolf-Ruediger; Schneider, Armin; Krueger, Carola; Sommer, Clemens; Schwab, Stefan; Kollmar, Rainer; Maurer, Martin; Weber, Daniela; Gassler, Nikolaus

PATENT ASSIGNEE(S): Axaron Bioscience Ag, Germany

SOURCE: U.S. Pat. Appl. Publ., 169 pp., Cont.-in-part of Appl. No. PCT/IB03/06446.

CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005142102	A1	20050630	US 2004-880101	20040630
US 2004141946	A1	20040722	US 2003-659295	20030911
WO 2004058287	A2	20040715	WO 2003-IB6446	20031231
WO 2004058287	A8	20041021		
WO 2004058287	A3	20041216		
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW			
RW:	BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
WO 2006008582	A1	20060126	WO 2004-IB4329	20041229
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW			
RW:	AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			

PRIORITY APPLN. INFO.: US 2002-331755 B1 20021231  
US 2003-659295 A2 20030911  
WO 2003-IB6446 A2 20031231  
US 2004-880101 A 20040630

AB The present invention relates to a method of treating a neurol. condition in a mammal by administering at least one hematopoietic growth factor from the group consisting of GCSF, GMCSF, IL-3, IL-5, a derivative thereof, or a mimetic thereof. A method is also claimed of treating a neurol. condition using neural stem cells treated with a hematopoietic factor. Also claimed is a method of enhancing the survival of a cell transplanted into a mammal, comprising introducing into the cell one or more polynucleotides which encode a hematopoietic factor.

A method of enhancing the viability of a neural cell culture comprising contacting the neural cell culture with a hematopoietic factor is addnl. claimed.

L5 ANSWER 5 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2005304965 EMBASE  
TITLE: Thrombocytosis during antifungal therapy of candidemia.  
AUTHOR: Saathoff A.D.; Elkins S.L.; Chapman S.W.; McAllister S.F.; Cleary J.D.  
CORPORATE SOURCE: Dr. J.D. Cleary, University of Mississippi Medical Center, 2500 N. State St., Jackson, MS 39216-4500, United States. Jcleary@umsmed.edu  
SOURCE: Annals of Pharmacotherapy, (2005) Vol. 39, No. 7-8, pp. 1238-1243. .  
Refs: 24  
ISSN: 1060-0280 CODEN: APHRER  
COUNTRY: United States  
DOCUMENT TYPE: Journal; Article  
FILE SEGMENT: 004 Microbiology  
006 Internal Medicine  
025 Hematology  
037 Drug Literature Index  
038 Adverse Reactions Titles  
LANGUAGE: English  
SUMMARY LANGUAGE: English; Spanish; French  
ENTRY DATE: Entered STN: 29 Sep 2005  
Last Updated on STN: 29 Sep 2005

AB BACKGROUND: Secondary, "reactive," thrombocytosis has been attributed to bacterial infection and treatment with multiple pharmaceuticals and may be associated with an increase in the incidence of gastrointestinal tract bleeding and thrombotic events (eg, stroke). OBJECTIVE: To characterize the dynamics of thrombocytosis in patients with candidemia receiving antifungal therapy. METHODS: We initiated a retrospective observational description of patients with candidemia who were treated with antifungal agents. A total of 108 patients diagnosed with candidemia between August 1995 and September 2003 at our teaching hospital were enrolled. Three groups (candidemia with antifungal therapy, candidemia without antifungal therapy, antifungal therapy without candidemia) of patients >18 years of age were evaluated for the presence of thrombocytosis. Platelet administration, pharmacologic or pathologic contributors to thrombocytosis, and other pertinent details related to an elevation of platelet counts were scrutinized. RESULTS: Reactive thrombocytosis was observed in approximately 10% of treated patients with candidemia. Within the subgroup developing reactive thrombocytosis, life-threatening thrombotic complications were uncommon. Mean baseline platelet counts were  $393 \times 10^3/\text{mm}^3$ , with a mean peak ( $695 \times 10^3/\text{mm}^3$ ) occurring an average of 13 days after initiation of therapy. All patients had resolution within 7 days after therapy. The maximum peak ( $1056 \times 10^3/\text{mm}^3$ ) was observed in a patient after 14 days of antifungal therapy. The onset of thrombocytosis in this patient was 4 days and lasted 4 days after therapy. CONCLUSIONS: Reactive thrombocytosis occurs during treatment of candidemia. The causative agent (drug vs disease), the risk associated with this reaction, and evaluation of treatment need to be elucidated by a larger epidemiologic study or controlled, prospective clinical trial.

L5 ANSWER 6 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2005561466 EMBASE  
TITLE: Angiogenesis in the human heart: Gene and cell therapy.  
AUTHOR: Tirziu D.; Simons M.  
CORPORATE SOURCE: M. Simons, Departments of Medicine and Pharmacology and Toxicology, Dartmouth Medical School, Dartmouth-Hitchcock

Medical Center, Lebanon, NH 03756, United States.  
michael.simons@dartmouth.edu  
SOURCE: Angiogenesis, (2005) Vol. 8, No. 3, pp. 241-251. .  
Refs: 113  
ISSN: 0969-6970 CODEN: AGIOFT  
COUNTRY: Netherlands  
DOCUMENT TYPE: Journal; General Review  
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery  
022 Human Genetics  
025 Hematology  
037 Drug Literature Index  
038 Adverse Reactions Titles  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
ENTRY DATE: Entered STN: 26 Jan 2006  
Last Updated on STN: 26 Jan 2006

AB The concept of therapeutic angiogenesis - stimulation of new vessels growth to restore blood supply to ischemic tissue has been studied in a number of clinical trials in patients with advanced coronary and peripheral arterial disease. This review discusses the main biological processes underlying new vessel growth and addresses applications of growth factor and cell therapy based on the stimulation of angiogenesis. While still very young and controversial, cell therapy has an enormous potential that is yet to be explored. Multiple questions remain unanswered including the choice of the best cell type, patient selection and the mechanism of action. Nevertheless, much should be expected in this area in the next decade with the likely emergence of new therapies for treatment of ischemic diseases. .COPYRGT. Springer 2005.

L5 ANSWER 7 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2004536240 EMBASE  
TITLE: Treatment of head and neck cancer in elderly patients: State of the art and guidelines.  
AUTHOR: Bernardi D.; Barzan L.; Franchin G.; Cinelli R.; Balestreri L.; Tirelli U.; Vaccher E.  
CORPORATE SOURCE: oma@cro.it  
SOURCE: Critical Reviews in Oncology/Hematology, (2005) Vol. 53, No. 1, pp. 71-80. .  
Refs: 48  
ISSN: 1040-8428 CODEN: CCRHEC  
PUBLISHER IDENT.: S 1040-8428(04)00136-2  
COUNTRY: Ireland  
DOCUMENT TYPE: Journal; General Review  
FILE SEGMENT: 014 Radiology  
016 Cancer  
037 Drug Literature Index  
038 Adverse Reactions Titles  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
ENTRY DATE: Entered STN: 6 Jan 2005  
Last Updated on STN: 6 Jan 2005

AB Although the majority of head and neck cancers occur between the fifth and sixth decade, their onset in patients older than 60 years is not a rare event. A peculiar characteristic of almost all case series is the lower prevalence of radical treatments among elderly as compared to younger patients, in particular surgery and combined treatment of surgery plus radiation therapy or chemotherapy and radiation therapy. Radiotherapy is a feasible treatment in elderly patients, also in very advanced age groups and, in the era of organ preservation, chemotherapy combined with RT has a paramount importance. Therapeutical planning must be based not only on tumor characteristics, but also on the physiological, rather than the chronological age the patient. The main clinical problem is, therefore, the selection of patients to be

administered anticancer treatment. In patients aged 70 or older, complete geriatric assessment and a multidisciplinary approach are the crucial points. .COPYRG.T. 2004 Elsevier Ireland Ltd. All rights reserved.

L5 ANSWER 8 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2005474679 EMBASE  
TITLE: [Stem cell therapy in patients with peripheral arterial occlusive disease].  
STAMMZELLTHERAPIE BEI PAVK.  
AUTHOR: Kopp Ch.W.; Steiner-Boker S.; Gschwandtner M.; Minar E.  
CORPORATE SOURCE: Dr. Ch.W. Kopp, Universitätsklinik für Innere Medizin II, Abteilung für Angiologie, Wahringer Gürtel 18-20, A-1090 Wien, Austria. Christoph.kopp@meduniwien.ac.at  
SOURCE: Zeitschrift für Gefäßmedizin, (2005) Vol. 2, No. 3, pp. 12-15.  
Refs: 27  
ISSN: 1812-9501  
COUNTRY: Austria  
DOCUMENT TYPE: Journal; General Review  
FILE SEGMENT: 006 Internal Medicine  
018 Cardiovascular Diseases and Cardiovascular Surgery  
021 Developmental Biology and Teratology  
029 Clinical Biochemistry  
LANGUAGE: German  
SUMMARY LANGUAGE: English; German  
ENTRY DATE: Entered STN: 8 Dec 2005  
Last Updated on STN: 8 Dec 2005

AB Autologous bone marrow (BM)-derived stem cell therapy for the induction of therapeutic angiogenesis is a potentially limb-saving strategy in patients with chronic limb ischemia and no surgical or interventional option for revascularisation. This review shall present the underlying therapeutic concept and clinical guidelines how to gain profit of the angiogenic potential of BM-derived stem cells. Finally, stem cell mobilization and targeted homing will be discussed as a potential alternative to BM-derived stem cell transplantation.

L5 ANSWER 9 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:565109 CAPLUS  
DOCUMENT NUMBER: 141:100449  
TITLE: Methods of treating neurological conditions with hematopoietic growth factors  
INVENTOR(S): Schaebitz, Wolf-Ruediger; Schneider, Armin; Krueger, Carola; Sommer, Clemens; Schwab, Stefan; Kollmar, Rainer; Maurer, Martin; Weber, Daniela; Gassler, Nikolaus  
PATENT ASSIGNEE(S): Axaron Bioscience AG, Germany  
SOURCE: PCT Int. Appl., 210 pp.  
CODEN: PIXXD2  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 2  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004058287	A2	20040715	WO 2003-IB6446	20031231
WO 2004058287	A8	20041021		
WO 2004058287	A3	20041216		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO,



NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ,  
 TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW  
 RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ,  
 BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE,  
 ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK,  
 TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2004141946 A1 20040722 US 2003-659295 20030911  
 CA 2511294 A1 20040715 CA 2003-2511294 20031231  
 AU 2003299430 A1 20040722 AU 2003-299430 20031231  
 EP 1581249 A2 20051005 EP 2003-799727 20031231

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,  
 IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK

BR 2003017910 A 20051129 BR 2003-17910 20031231  
 CN 1756556 A 20060405 CN 2003-80110075 20031231  
 JP 2006512419 T 20060413 JP 2005-509731 20031231  
 US 2005142102 A1 20050630 US 2004-880101 20040630

PRIORITY APPLN. INFO.:

US 2002-331755 A 20021231  
 US 2003-659295 A 20030911  
 WO 2003-IB6446 W 20031231

AB The present invention relates to a method of treating neurol.  
 conditions in a mammal by administering a hematopoietic growth factor such  
 as granulocyte-colony stimulating factor (GCSF) and granulocyte-  
 macrophage colony stimulating factor  
 (GMCSF). The invention also provides methods of screening for  
 compds. that bind to a GCSF or GMCSF receptor found on the  
 surface of a neuronal cell; and which provides a neuroprotective,  
 neuroproliferative and/or a STAT gene activation activity.

L5 ANSWER 10 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:162774 CAPLUS  
 DOCUMENT NUMBER: 140:210821  
 TITLE: Cell modulation using a cytoskeletal protein  
 INVENTOR(S): Losordo, Douglas W.; Kishore, Raj  
 PATENT ASSIGNEE(S): Caritas St. Elizabeth's Medical Center of Boston,  
 Inc., USA  
 SOURCE: PCT Int. Appl., 79 pp.  
 CODEN: PIXXD2  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 1  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004016739	A2	20040226	WO 2003-US23978	20030801
WO 2004016739	A3	20050414		
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW			
RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
AU 2003257960	A1	20040303	AU 2003-257960	20030801
US 2004105860	A1	20040603	US 2003-633407	20030801
PRIORITY APPLN. INFO.:			US 2002-400084P	P 20020801
			WO 2003-US23978	W 20030801

AB Disclosed are compns. and methods for modulating endothelial cells (ECs)  
 in a mammal. Practice of the invention generally involves changing  
 activity of the ezrin cytoskeletal protein sufficient to increase or  
 decrease proliferation of the cells. Also disclosed are useful screens

for detecting agents capable of modulating ezrin activity. The invention has a variety of useful applications including use in the treatment of diseases associated with unsatisfactory EC proliferation.

L5 ANSWER 11 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN  
 ACCESSION NUMBER: 2004:117103 CAPLUS  
 DOCUMENT NUMBER: 140:157937  
 TITLE: Use of erythropoietin  
 PATENT ASSIGNEE(S): Bahlmann, Ferdinand Hermann, Germany  
 SOURCE: Ger. Offen., 17 pp.  
 CODEN: GWXXBX  
 DOCUMENT TYPE: Patent  
 LANGUAGE: German  
 FAMILY ACC. NUM. COUNT: 1  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 10234192	A1	20040212	DE 2002-10234192	20020726
CA 2493598	A1	20040212	CA 2003-2493598	20030725
WO 2004012759	A2	20040212	WO 2003-EP8229	20030725
WO 2004012759	A3	20040603		
WO 2004012759	B1	20040708		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003255290	A1	20040223	AU 2003-255290	20030725
EP 1526867	A2	20050504	EP 2003-766302	20030725
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
BR 2003012981	A	20050614	BR 2003-12981	20030725
CN 1681526	A	20051012	CN 2003-822116	20030725
JP 2006503001	T	20060126	JP 2004-525322	20030725
NO 2005001002	A	20050418	NO 2005-1002	20050224
US 2005272634	A1	20051208	US 2005-522426	20050325
PRIORITY APPLN. INFO.:			DE 2002-10234192	A 20020726
			WO 2003-EP8229	W 20030725

AB The present invention concerns the use of erythropoietin for stimulation of physiol. mobilization, proliferation and differentiation of endothelial progenitor cells, for stimulation of angiogenesis, for therapy of diseases connected to a dysfunction of endothelial progenitor cells, and for production of pharmaceutical compns. for treatment of such diseases as well as pharmaceutical compns., which contain erythropoietin and other suitable active substances for stimulation of endothelial progenitor cells.

L5 ANSWER 12 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN  
 ACCESSION NUMBER: 2005021293 EMBASE  
 TITLE: [Pathophysiology and therapy of reversible posterior leukoencephalopathy syndrome (RPLS)].  
 PATHOPHYSIOLOGIE UND THERAPIE DES REVERSIBLEN POSTERIOREN LEUKOENZEPHALOPATHIESYNDROMS (RPLS).  
 AUTHOR: Obermann M.; Kastrup O.; Glzewski E.; Maschke M.  
 CORPORATE SOURCE: M. Obermann, Universitätsklinikum Essen, Klin. und Poliklin. F. Neurologie, Hufelandstrasse 55, 45122 Essen,

Germany. mark.obermann@uni-essen.de  
SOURCE: Aktuelle Neurologie, (2004) Vol. 31, No. 10, pp. 481-489. .  
Refs: 75  
ISSN: 0302-4350 CODEN: AKNUAR  
COUNTRY: Germany  
DOCUMENT TYPE: Journal; General Review  
FILE SEGMENT: 008 Neurology and Neurosurgery  
026 Immunology, Serology and Transplantation  
037 Drug Literature Index  
LANGUAGE: German  
SUMMARY LANGUAGE: English; German  
ENTRY DATE: Entered STN: 20 Jan 2005  
Last Updated on STN: 20 Jan 2005

AB Reversible posterior leukoencephalopathy syndrome (RPLS) is a widely recognized neurological disorder, considering the increasing number of publications over the past two years. Oedematous cerebral white matter lesions particularly involve the posterior parietal and occipital lobes, but may also affect the brainstem, basal ganglia and cerebellum. This leads to characteristic neurological symptoms such as headache, visual disturbances, nausea and vomiting, altered mental status and seizures. This syndrome is often associated with an abrupt increase in blood pressure, mainly in patients with eclampsia, renal insufficiency and hypertensive encephalopathy. Immunosuppressive and immunomodulating drugs such as cyclosporine A, tacrolimus, interferone- $\alpha$  and filgastim may also lead to RPLS. A rare variant of RPLS is the isolated brainstem leukoencephalopathy, which is characterized by extensive MRI lesions associated with little clinical symptoms. The respective lesions are best visualized with FLAIR and T(2)-weighted magnetic resonance imaging. They show diffuse hyperintensity generally involving cerebral parieto-occipital regions, but may also selectively affect the brainstem without accompanying supratentorial lesions. Diffusion weighted imaging is an important diagnostic tool to differentiate the mainly vasogenic edema of RPLS from the cytotoxic edema of acute cerebral ischaemia. Appropriate therapy consists of rapid and sustained correction of hypertension. Symptoms can be completely reversible and MRI lesions may show complete remission. Early recognition of RPLS is extremely important, because of its benign prognosis under therapy. Delay of appropriate treatment, however, may lead to permanent damage of affected brain tissue. This review will give an overview of current knowledge of pathophysiology, diagnostic procedures and treatment options on RPLS. Special consideration will be given to reversible isolated brainstem leukoencephalopathy.

L5 ANSWER 13 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2004117498 EMBASE  
TITLE: Perspectives for gene therapy in renal diseases.  
AUTHOR: Imai E.; Isaka Y.  
CORPORATE SOURCE: Dr. E. Imai, Division of Nephrology, Department of Internal Medicine, Osaka Univ. Grad. Sch. of Med., Suita, Osaka 565-0871, Japan  
SOURCE: Internal Medicine, (2004) Vol. 43, No. 2, pp. 85-96. .  
Refs: 92  
ISSN: 0918-2918 CODEN: IEDIEP  
COUNTRY: Japan  
DOCUMENT TYPE: Journal; General Review  
FILE SEGMENT: 022 Human Genetics  
028 Urology and Nephrology  
037 Drug Literature Index  
038 Adverse Reactions Titles  
039 Pharmacy  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
ENTRY DATE: Entered STN: 25 Mar 2004

Last Updated on STN: 25 Mar 2004

AB Somatic cell gene therapy has made considerable progress last five years and has shown clear success in some clinical trials. In the field of nephrology, both the elucidation of pathophysiology of renal diseases and the development of gene transfer technique have become driving force for new therapy of incurable renal diseases, such as Alport syndrome and polycystic kidney disease. Gene therapy of renal cancer, although its application is limited to advanced cancer, is the front-runner of clinical application. Erythropoietin gene therapy has provided encouraging results for the treatment of anemia in uremic rats and recently progressed to the inducible one in response to hypoxia. Gene therapy for glomerulonephritis and renal fibrosis showed prominent impact on experimental models, although the safety must be confirmed for prolonged treatment. Transplant kidney is an ideal material for gene modification and induction of tolerance in the transplant kidney is an attractive challenge. Emerging techniques are becoming available such as stem cell technology and messenger RNA silencing strategies. We believe that the future of gene therapy research is exciting and promising and it holds an enormous potential for clinical application.

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ACCESSION NUMBER: 2005043742 EMBASE

TITLE: Generalisability in economic evaluation studies in healthcare: A review and case studies.

AUTHOR: Sculpher M.J.; Pang F.S.; Manca A.; Drummond M.F.; Golder S.; Urdahl H.; Davies L.M.; Eastwood A.

CORPORATE SOURCE: M.J. Sculpher, Centre for Health Economics, University of York, York, United Kingdom

SOURCE: Health Technology Assessment, (2004) Vol. 8, No. 49, pp. iii-117.  
Refs: 286

ISSN: 1366-5278 CODEN: HTASFX

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 004 Microbiology  
018 Cardiovascular Diseases and Cardiovascular Surgery  
033 Orthopedic Surgery  
036 Health Policy, Economics and Management  
037 Drug Literature Index  
038 Adverse Reactions Titles

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 10 Feb 2005

Last Updated on STN: 10 Feb 2005

AB Objectives: To review, and to develop further, the methods used to assess and to increase the generalisability of economic evaluation studies. Data sources: Electronic databases. Review methods: Methodological studies relating to economic evaluation in healthcare were searched. This included electronic searches of a range of databases, including PREMEDLINE, MEDLINE, EMBASE and EconLit, and manual searches of key journals. The case studies of a decision analytic model involved highlighting specific features of previously published economic studies related to generalisability and location-related variability. The case-study involving the secondary analysis of cost-effectiveness analyses was based on the secondary analysis of three economic studies using data from randomised trials. Results: The factor most frequently cited as generating variability in economic results between locations was the unit costs associated with particular resources. In the context of studies based on the analysis of patient-level data, regression analysis has been advocated as a means of looking at variability in economic results across locations. These methods have generally accepted that some components of resource use and outcomes are exchangeable across locations. Recent

studies have also explored, in cost-effectiveness analysis, the use of tests of heterogeneity similar to those used in clinical evaluation in trials. The decision analytic model has been the main means by which cost-effectiveness has been adapted from trial to non-trial locations. Most models have focused on changes to the cost side of the analysis, but it is clear that the effectiveness side may also need to be adapted between locations. There have been weaknesses in some aspects of the reporting in applied cost-effectiveness studies. These may limit decision-makers' ability to judge the relevance of a study to their specific situations. The case study demonstrated the potential value of multilevel modelling (MLM). Where clustering exists by location (e.g. centre or country), MLM can facilitate correct estimates of the uncertainty in cost-effectiveness results, and also a means of estimating location-specific cost-effectiveness. The review of applied economic studies based on decision analytic models showed that few studies were explicit about their target decision-maker(s)/jurisdictions. The studies in the review generally made more effort to ensure that their cost inputs were specific to their target jurisdiction than their effectiveness parameters. Standard sensitivity analysis was the main way of dealing with uncertainty in the models, although few studies looked explicitly at variability between locations. The modelling case study illustrated how effectiveness and cost data can be made location-specific. In particular, on the effectiveness side, the example showed the separation of location-specific baseline events and pooled estimates of relative treatment effect, where the latter are assumed exchangeable across locations. Conclusions: A large number of factors are mentioned in the literature that might be expected to generate variation in the cost-effectiveness of healthcare interventions across locations. Several papers have demonstrated differences in the volume and cost of resource use between locations, but few studies have looked at variability in outcomes. In applied trial-based cost-effectiveness studies, few studies provide sufficient evidence for decision-makers to establish the relevance or to adjust the results of the study to their location of interest. Very few studies utilised statistical methods formally to assess the variability in results between locations. In applied economic studies based on decision models, most studies either stated their target decision-maker/jurisdiction or provided sufficient information from which this could be inferred. There was a greater tendency to ensure that cost inputs were specific to the target jurisdiction than clinical parameters. Methods to assess generalisability and variability in economic evaluation studies have been discussed extensively in the literature relating to both trial-based and modelling studies. Regression-based methods are likely to offer a systematic approach to quantifying variability in patient-level data. In particular, MLM has the potential to facilitate estimates of cost-effectiveness, which both reflect the variation in costs and outcomes between locations and also enable the consistency of cost-effectiveness estimates between locations to be assessed directly. Decision analytic models will retain an important role in adapting the results of cost-effectiveness studies between locations. Recommendations for further research include: the development of methods of evidence synthesis which model the exchangeability of data across locations and allow for the additional uncertainty in this process; assessment of alternative approaches to specifying multilevel models to the analysis of cost-effectiveness data alongside multilocation randomised trials; identification of a range of appropriate covariates relating to locations (e.g. hospitals) in multilevel models; and further assessment of the role of econometric methods (e.g. selection models) for cost-effectiveness analysis alongside observational datasets, and to increase the generalisability of randomised trials. .COPYRGT. Queen's Printer and Controller of HMSO 2004. All rights reserved.